STIC Search Report
Biotech-Chem Library

STIC Database Tracking Number: 155626

TO: Ralph J Gitomer **Location: rem/3D65/3E71** **Art Unit: 1651** **Friday, June 10, 2005** **Case Serial Number: 10/615966**	**From: Noble Jarrell** **Location: Biotech-Chem Library** **Rem 1B71** **Phone: 272-2556** **Noble.jarrell@uspto.gov**

Search Notes

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     (FILE 'HOME' ENTERED AT 07:50:20 ON 10 JUN 2005)

     FILE 'HCAPLUS' ENTERED AT 07:50:59 ON 10 JUN 2005
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L1           26 SEA ABB=ON  PLU=ON  ("WATKINS S"/AU OR "WATKINS S M"/AU)
                E WATKINS STEVEN/AU
L2           24 SEA ABB=ON  PLU=ON  ("WATKINS STEVEN"/AU OR "WATKINS STEVEN
                M"/AU OR "WATKINS STEVEN MICHAEL"/AU)
                E WATKINS STEPHEN/AU
L3           10 SEA ABB=ON  PLU=ON  ("WATKINS STEPHEN"/AU OR "WATKINS STEVE"/AU
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L4           12 SEA ABB=ON  PLU=ON  LIPOMICS/CS,PA
L5            1 SEA ABB=ON  PLU=ON  (US2002-436192# OR US2002-424949# OR
                US2002-401684# OR US2002-373912# OR US2002-363587#)/AP,PRN

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L6              TRA L5 1- RN :       20 TERMS

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L5   ANSWER 1 OF 1  HCAPLUS  COPYRIGHT 2005 ACS on STN
AN   2003:757811  HCAPLUS
DN   139:271092
ED   Entered STN:  26 Sep 2003
TI   Novel metabolic targets and markers
IN   Watkins, Steven M.; Baillie, Rebecca A.
PA   Lipomics Technologies, Inc., USA
SO   PCT Int. Appl., 60 pp.
     CODEN: PIXXD2
DT   Patent
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LA English
IC ICM C12N
CC 1-12 (Pharmacology)
Section cross-reference(s): 2, 9
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2003078574	A2	20030925	WO 2003-US7242	20030307 <--
	WO 2003078574	A3	20040219		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

	CA 2477909	AA	20030925	CA 2003-2477909	20030307 <--
	US 2004024065	A1	20040205	US 2003-383850	20030307 <--
	EP 1490076	A2	20041229	EP 2003-744631	20030307 <--

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK

PRAI	US 2002-363587P	P	20020311 <--
	US 2002-373912P	P	20020419 <--
	US 2002-401684P	P	20020806 <--
	US 2002-424949P	P	20021108 <--
	US 2002-436192P	P	20021224 <--
	WO 2003-US7242	W	20030307

CLASS

PATENT NO.	CLASS	PATENT FAMILY CLASSIFICATION CODES
WO 2003078574	ICM	C12N
WO 2003078574	ECLA	A61K031/00; A61K031/51; A61K031/519; A61K038/22; A61K038/27; C12N009/10A <--
US 2004024065	NCL	514/560.000
	ECLA	A61K031/00; A61K031/51; A61K031/519; A61K038/22; A61K038/27; C12N009/10A <--

AB The present invention is based, in part, on the discovery that certain metabolites or metabolic pathways can be used as diagnostic or therapeutic markers. For example, phosphatidylethanolamine-N-methyltransferase (PEMT) activity and other metabolic activities or markers associated therewith can be used either as markers for diagnosing various conditions or as targets for therapeutic treatment of various disease conditions. In one embodiment, the present invention provides a method for regulating the level of a fatty acid in a system. The method includes decreasing the CDP-choline activity in the system. In still another embodiment, the present invention provides a method for regulating a lipoprotein component ratio in a system. The method includes regulating the PEMT activity in the system whereby regulating the lipoprotein component ratio in the system, wherein the lipoprotein component ratio is selected from the group consisting of cholesterol ester to phosphatidylcholine, cholesterol ester to apoprotein, free cholesterol to apoprotein, and triacylglyceride to phosphatidylcholine. In another embodiment, the present invention provides a method of assessing the d. of a lipoprotein in a system. In yet another embodiment, the present invention provides a method for treating or preventing a cardiovascular or neurol. condition.

ST phosphatidylcholine methyltransferase metabolic pathway disease treatment

IT Brain, disease
(Alpers' disease; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Congenital malformations
(Arnold-Chiari Malformation; phosphatidylethanolamine-N-

methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Mental disorder
(Asperger's Syndrome; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Brain, disease
(Gilles de la Tourette syndrome; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Brain
Heart
Intestine
Liver
Mammary gland
(PEMT of; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Mental disorder
(Pick's disease; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Brain, disease
(Rett syndrome; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Disease, animal
(Shy-Drager syndrome; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Nuclear receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(agonists, antitumor activity of; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Antiarteriosclerotics
(antiatherosclerotics; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Estrogens
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(antiestrogens, infertility from; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Peroxisome proliferators
(antitumor activity of; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Heart, disease
(arrhythmia; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Eicosanoids
Thyroid hormones

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(assessment of; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Allergy
(atopy, congenital malformations from; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Heart, disease
(attack; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Mental disorder
(attention deficit hyperactivity disorder; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Mental disorder
(autism; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Brain, disease
(calcinosis; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Ischemia
(cardiac; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Heart, disease
(cardiomyopathy; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Movement disorders
(cerebral palsy; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Paralysis
(childhood alternating hemiplegia; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Apolipoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(cholesterol esters; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Intestine, neoplasm
(colon; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Diabetes mellitus
(congenital malformations from; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Artery, disease
(coronary; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Sperm
(decreased motility; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Nerve, disease
(degeneration; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Bone, disease
(demineralization; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Mental disorder
(diffuse Lewy body disease; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Calcification
(disorder, calcinosis, cerebral; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Immunity
(disorder, congenital malformations from; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Carcinoma
(esophageal squamous cell; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Kidney, disease
(failure; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Blood plasma
(fatty acids and lipoproteins of; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Animal tissue
(fatty acids of; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Phosphatidylcholines, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(fatty acids of; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Liver, disease
(fatty; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Embryo, animal
(fetus, treatment of; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Lipoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(high-d.; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Head, disease
(holoprosencephaly; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Brain, disease
(hydranencephaly; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Brain, disease
(hydrocephalus, Dandy Walker Syndrome; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Lipids, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(hyperlipidemia; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Milk
(increase in production of; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Development, mammalian postnatal
(infant, treatment of; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Human immunodeficiency virus
(infection, parenterally acquired; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Intestine, disease
(inflammatory; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Lipoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(intermediate-d.; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Heart, disease
(ischemia; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Neuronal ceroid lipofuscinosis
(juvenile; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and

lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Transplant and Transplantation
(liver; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Lipoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(low-d.; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Fertility
(male, disorder; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Head, disease
(microcephaly; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Nerve, disease
(motor; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Nervous system, disease
(multiple system atrophy; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Mental disorder
(neurotic depression; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Glycerides, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(of phosphatidylcholines; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT AIDS (disease)
(parenterally acquired; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Alzheimer's disease
Anti-Alzheimer's agents
Anti-inflammatory agents
Antiarrhythmics
Anticholesteremic agents
Antihypertensives
Antiobesity agents
Antiparkinsonian agents
Antirheumatic agents
Antitumor agents
Atherosclerosis
Autoimmune disease
Cardiovascular agents
Cardiovascular system, disease
Chylomicrons
Cirrhosis
Cockayne's syndrome

Congenital malformations
Diagnosis
Fetal alcohol syndrome
Fragile X syndrome
Human
Hypercholesterolemia
Hyperglycemia
Hypertension
Hyperthyroidism
Hypertriglyceridemia
Hypolipemic agents
Hypothyroidism
Infection
Inflammation
Neoplasm
Nervous system, disease
Obesity
Osteoporosis
Pancreas, neoplasm
Parkinson's disease
Phenylketonuria
Poliomyelitis
Prostate gland, neoplasm
Rheumatoid arthritis
Skin, disease
Teratogenesis
Thyroid gland, neoplasm
(phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Fatty acids, biological studies
Lipids, biological studies
Lipoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Estrogens
Sex hormones
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Fatty acids, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(polyunsatd.; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Paralysis
(pseudobulbar; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Fatty acids, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(saturated; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Spinal column, disease
(spina bifida; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT Nervous system, disease
(spinocerebellar ataxia; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Esophagus, neoplasm
(squamous cell carcinoma; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Brain, disease
(stroke; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Heart
Liver
(toxicity, PEMT of; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Liver
(transplant; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Lactation
(treatment in; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Brain, disease
(tuberous sclerosis; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Lipoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(very-low-d.; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Infection
(viral, HIV, parenterally acquired; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Adrenoceptor agonists
(β1-; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Adrenoceptor agonists
(β2-; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT Adrenoceptor agonists
(β3-; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)
IT 9004-10-8, Insulin, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(assessment of; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and

lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT 6027-13-0, Homocysteine
RL: BSU (Biological study, unclassified); BIOL (Biological study) (in lipoproteins determination; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT 262-12-4D, Dibenzo[b,e][1,4]dioxin, chloro derivs.
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study) (infertility from; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT 57-88-5, Cholesterol, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study) (of apolipoproteins; phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT 57-10-3, Hexadecanoic acid, biological studies 57-11-4, Octadecanoic acid, biological studies 57-88-5D, Cholesterol, esters 60-33-3, 9,12-Octadecadienoic acid (9Z,12Z)-, biological studies 373-49-9 506-17-2 506-32-1 979-92-0, S-Adenosylhomocysteine 987-78-0, CDP-choline 6217-54-5 10417-94-4 37256-91-0, Phosphatidylethanolamine-N-methyltransferase
RL: BSU (Biological study, unclassified); BIOL (Biological study) (phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT 9002-72-6, Growth hormone 29908-03-0
RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses) (phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

IT 59-30-3, Folic acid, biological studies 59-67-6, Niacin, biological studies 8059-24-3, Vitamin B6
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses) (phosphatidylethanolamine-N-methyltransferase (PEMT) as metabolic target and marker to regulate fatty acid levels and lipoprotein component ratio for disease treatment in relation to CDP-choline)

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TSCA INFORMATION NOW CURRENT THROUGH JANUARY 18, 2005

Please note that search-term pricing does apply when conducting SmartSELECT searches.

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*                                                                    *
* The CA roles and document type information have been removed from *
* the IDE default display format and the ED field has been added,   *
* effective March 20, 2005.  A new display format, IDERL, is now    *
* available and contains the CA role and document type information. *
*                                                                    *
**********************************************************************

Crossover limits have been increased.  See HELP CROSSOVER for details.

Experimental and calculated property data are now available. For more
information enter HELP PROP at an arrow prompt in the file or refer
to the file summary sheet on the web at:
http://www.cas.org/ONLINE/DBSS/registryss.html

=> d ide l7 tot


L7   ANSWER 1 OF 20  REGISTRY  COPYRIGHT 2005 ACS on STN
RN   37256-91-0  REGISTRY
ED   Entered STN:  16 Nov 1984
CN   Methyltransferase, phosphatidylethanolamine (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   E.C. 2.1.1.17
CN   Lipid methyl transferase
CN   Phosphatidylethanolamine methyltransferase
CN   Phosphatidylethanolamine N-methyltransferase
CN   Phosphatidylethanolamine-N-methylase
CN   Phosphatidylethanolamine-S-adenosylmethionine methyltransferase
CN   S-Adenosylmethionine-phosphatidylethanolamine methyltransferase
DR   9068-27-3
MF   Unspecified
CI   MAN
LC   STN Files:   AGRICOLA, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CAPLUS, CIN,
       EMBASE, TOXCENTER, USPATFULL

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
             322 REFERENCES IN FILE CA (1907 TO DATE)
             322 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L7   ANSWER 2 OF 20  REGISTRY  COPYRIGHT 2005 ACS on STN
RN   29908-03-0  REGISTRY
ED   Entered STN:  16 Nov 1984
CN   Adenosine, 5'-[[(3S)-3-amino-3-carboxypropyl]methylsulfonio]-5'-deoxy-,
     inner salt (9CI)  (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   Adenosine, 5'-[(3-amino-3-carboxypropyl)methylsulfonio]-5'-deoxy-,
     hydroxide, inner salt, (3S)-
CN   Adenosine, 5'-[(L-3-amino-3-carboxypropyl)methylsulfonio]-5'-deoxy-,
     hydroxide, inner salt (8CI)
CN   Methionine, S-adenosyl- (6CI)
OTHER NAMES:
CN   Active methionine
CN   Ademetionine
CN   AdoMet
CN   Donamet
CN   L-Methionine, S-adenosyl-
CN   L-S-Adenosylmethionine
CN   S Amet
CN   S-Adenosyl-L-methionine
CN   SAMe
FS   STEREOSEARCH
DR   23095-97-8, 2613-02-7, 86522-35-2, 86866-89-9, 5134-37-2, 28378-99-6
MF   C15 H22 N6 O5 S
CI   COM
LC   STN Files:   ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS,
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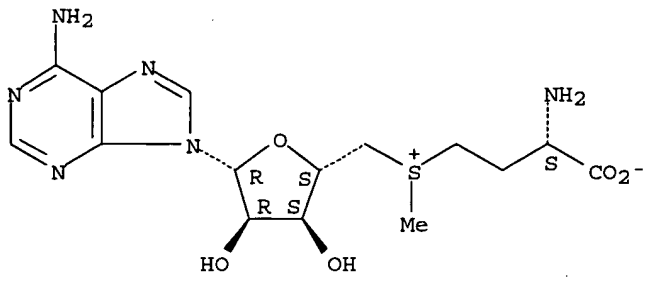
BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, DDFU, DRUGU, EMBASE, IFICDB, IFIPAT, IFIUDB, IMSRESEARCH, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PIRA, PROMT, PS, RTECS*, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

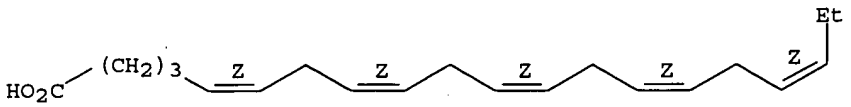
Absolute stereochemistry.



3656 REFERENCES IN FILE CA (1907 TO DATE).
111 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
3659 REFERENCES IN FILE CAPLUS (1907 TO DATE)
51 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L7 ANSWER 3 OF 20 REGISTRY COPYRIGHT 2005 ACS on STN
RN **10417-94-4** REGISTRY
ED Entered STN: 16 Nov 1984
CN 5,8,11,14,17-Eicosapentaenoic acid, (5Z,8Z,11Z,14Z,17Z)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 5,8,11,14,17-Eicosapentaenoic acid (6CI)
CN 5,8,11,14,17-Eicosapentaenoic acid, (all-Z)- (8CI)
OTHER NAMES:
CN (5Z,8Z,11Z,14Z,17Z)-Eicosapentaenoic acid
CN (all-cis)-5,8,11,14,17-Eicosapentaenoic acid
CN (all-Z)-Δ5,8,11,14,17-Eicosapentaenoic acid
CN (all-Z)-5,8,11,14,17-Eicosapentaenoic acid
CN Eicosapentaenoic acid
CN EPA
CN Icosapent
CN Icosapentaenoic acid
CN Timnodonic acid
FS STEREOSEARCH
DR 25377-48-4
MF C20 H30 O2
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CAOLD, CAPLUS, CASREACT, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, DDFU, DRUGU, EMBASE, IFICDB, IFIUDB, IMSDRUGNEWS, IMSRESEARCH, MRCK*, PATDPASPC, PHAR, PROMT, TOXCENTER, USAN, USPAT2, USPATFULL, VETU
(*File contains numerically searchable property data)
Other Sources: WHO

Double bond geometry as shown.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

8542 REFERENCES IN FILE CA (1907 TO DATE)
193 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
8554 REFERENCES IN FILE CAPLUS (1907 TO DATE)
6 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L7 ANSWER 4 OF 20 REGISTRY COPYRIGHT 2005 ACS on STN
RN **9004-10-8** REGISTRY
ED Entered STN: 16 Nov 1984
CN Insulin (9CI) (CA INDEX NAME)
OTHER NAMES:
CN Actrapid
CN Actrapid HM
CN Actrapid MC
CN Decurvon
CN Dermulin
CN Endopancrine
CN Exubera
CN HMR 4006
CN Iletin
CN Insular
CN Insulin Injection
CN Insulyl
CN Intesulin B
CN Iszilin
CN Mixtard
CN Musulin
DR 8049-67-0, 8049-95-4, 9004-12-0, 9037-76-7, 9045-63-0, 9045-65-2, 9045-66-3, 9045-67-4, 9066-39-1, 9066-40-4, 11081-38-2, 57126-42-8, 37243-75-7, 37294-43-2, 69090-47-7, 88026-11-3, 88026-12-4
MF Unspecified
CI PMS, COM, MAN
PCT Manual registration
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DIOGENES, DRUGU, EMBASE, HSDB*, IFICDB, IFIPAT, IFIUDB, IMSCOSEARCH, IPA, MEDLINE, MRCK*, NAPRALERT, NIOSHTIC, PDLCOM*, PHAR, PIRA, PROMT, RTECS*, TOXCENTER, USAN, USPAT2, USPATFULL, VTB
(*File contains numerically searchable property data)
Other Sources: EINECS**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

103300 REFERENCES IN FILE CA (1907 TO DATE)
1895 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
103374 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L7 ANSWER 5 OF 20 REGISTRY COPYRIGHT 2005 ACS on STN
RN **9002-72-6** REGISTRY
ED Entered STN: 16 Nov 1984
CN Somatotropin (9CI) (CA INDEX NAME)
OTHER NAMES:
CN Adenohypophyseal growth hormone
CN Anterior hypophyseal growth hormone
CN Anterior pituitary growth hormone
CN GH
CN Growth hormone
CN Hypophyseal growth hormone
CN Phyol
CN Phyone
CN Pituitary growth hormone

CN SH
CN Somacton
CN Somatotropic hormone
CN Sotropin H
CN STH
DR 9042-17-5, 9061-43-2, 9067-08-7
MF Unspecified
CI PMS, COM, MAN
PCT Manual registration
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAPLUS, CBNB, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DIOGENES, DRUGU, EMBASE, IFICDB, IFIPAT, IFIUDB, MEDLINE, MRCK*, NAPRALERT, NIOSHTIC, PIRA, PROMT, RTECS*, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

32468 REFERENCES IN FILE CA (1907 TO DATE)
617 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
32490 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L7 ANSWER 6 OF 20 REGISTRY COPYRIGHT 2005 ACS on STN
RN **8059-24-3** REGISTRY
ED Entered STN: 16 Nov 1984
CN Vitamin B6 (8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN Adermine
CN Vitamin H
DR 12001-78-4
MF Unspecified
CI COM, MAN
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMLIST, CIN, CSNB, DIOGENES, EMBASE, IFICDB, IFIPAT, IFIUDB, MEDLINE, NIOSHTIC, PROMT, TOXCENTER, USPAT2, USPATFULL
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

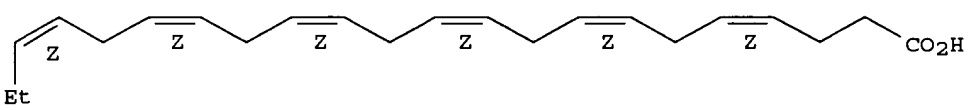
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

8931 REFERENCES IN FILE CA (1907 TO DATE)
175 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
8939 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L7 ANSWER 7 OF 20 REGISTRY COPYRIGHT 2005 ACS on STN
RN **6217-54-5** REGISTRY
ED Entered STN: 16 Nov 1984
CN 4,7,10,13,16,19-Docosahexaenoic acid, (4Z,7Z,10Z,13Z,16Z,19Z)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 4,7,10,13,16,19-Docosahexaenoic acid, (all-Z)- (8CI)
CN Docosahexaenoic acid (6CI)
OTHER NAMES:
CN (4Z,7Z,10Z,13Z,16Z,19Z)-4,7,10,13,16,19-Docosahexaenoic acid
CN (4Z,7Z,10Z,13Z,16Z,19Z)-Docosahexaenoic acid
CN (4Z,7Z,10Z,13Z,16Z,19Z)-Docosahexenoic acid
CN (all-Z)-4,7,10,13,16,19-Docosahexaenoic acid
CN Δ4,7,10,13,16,19-Docosahexaenoic acid
CN 4-cis,7-cis,10-cis,13-cis,16-cis,19-cis-Docosahexaenoic acid

CN all-cis-4,7,10,13,16,19-Docosahexaenoic acid
CN all-Z-Docosahexaenoic acid
CN Cervonic acid
CN DHA
CN Doconexent
CN Marinol D 50TG
FS STEREOSEARCH
DR 25377-50-8
MF C22 H32 O2
CI COM
LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CAOLD, CAPLUS, CASREACT, CEN, CHEMCATS, CIN, CSCHEM, EMBASE, IMSRESEARCH, MRCK*, PATDPASPC, PROMT, SYNTHLINE, TOXCENTER, USAN, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: WHO

Double bond geometry as shown.

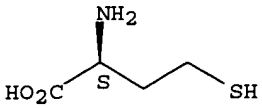


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

9074 REFERENCES IN FILE CA (1907 TO DATE)
158 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
9084 REFERENCES IN FILE CAPLUS (1907 TO DATE)
9 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L7 ANSWER 8 OF 20 REGISTRY COPYRIGHT 2005 ACS on STN
RN **6027-13-0** REGISTRY
ED Entered STN: 16 Nov 1984
CN L-Homocysteine (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Butyric acid, 2-amino-4-mercapto-, L- (8CI)
OTHER NAMES:
CN (S)-2-Amino-4-mercaptobutanoic acid
CN (S)-Homocysteine
CN 2-Amino-4-mercapto-L-butyric acid
CN 2-Amino-4-mercaptobutyric acid
CN Butanoic acid, 2-amino-4-mercapto-, (S)-
CN Homocysteine
CN NSC 43117
FS STEREOSEARCH
DR 454-28-4, 1867-00-1
MF C4 H9 N O2 S
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, DDFU, DIOGENES, DRUGU, EMBASE, GMELIN*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, PIRA, PROMT, RTECS*, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



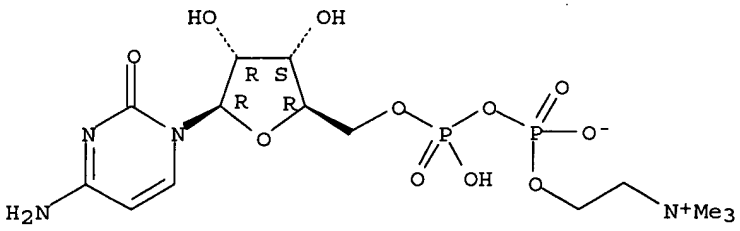
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

5572 REFERENCES IN FILE CA (1907 TO DATE)
104 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
5579 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L7 ANSWER 9 OF 20 REGISTRY COPYRIGHT 2005 ACS on STN
RN **987-78-0** REGISTRY
ED Entered STN: 16 Nov 1984
CN Cytidine 5'-(trihydrogen diphosphate), P'-[2-(trimethylammonio)ethyl] ester, inner salt (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Choline, hydroxide, 5'-ester with cytidine 5'-(trihydrogen pyrophosphate), inner salt (8CI)
CN Cytidine 5'-(trihydrogen diphosphate), P'-[2-(trimethylammonio)ethyl] ester, hydroxide, inner salt
OTHER NAMES:
CN Audes
CN CDP-choline
CN Cereb
CN Choline 5'-cytidine diphosphate
CN Choline cytidine diphosphate
CN Citicholine
CN Citicoline
CN Citidoline
CN Citifar
CN Colite
CN Corenalin
CN Cyscholin
CN Cytidine 5'-(choline diphosphate)
CN Cytidine 5'-(cholinyl pyrophosphate)
CN Cytidine 5'-diphosphate choline
CN Cytidine 5'-diphosphocholine
CN Cytidine choline diphosphate
CN Cytidine diphosphate choline
CN Cytidine diphosphate choline ester
CN Cytidine diphosphocholine
CN Cytidine diphosphorylcholine
CN Cytidoline
CN Difosfocin
CN Emicholine F
CN Ensign
CN Haocolin
CN Hornbest
CN Neucolis
CN Nicholin
CN Nicolin
CN Niticolin
CN NSC 122002
CN Reagin
CN Recofnan
CN Recognan
CN Rexort
CN Sintoclar
CN Somazina
CN Somazine
CN Suncholin
FS STEREOSEARCH

DR 769081-33-6, 1477-47-0, 64143-42-6
MF C14 H26 N4 O11 P2
CI COM
LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, DDFU, DRUGU, EMBASE, IMSCOSEARCH, IMSDRUGNEWS, IMSRESEARCH, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PHAR, PROMT, PROUSDDR, PS, RTECS*, TOXCENTER, USAN, USPAT2,. USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.

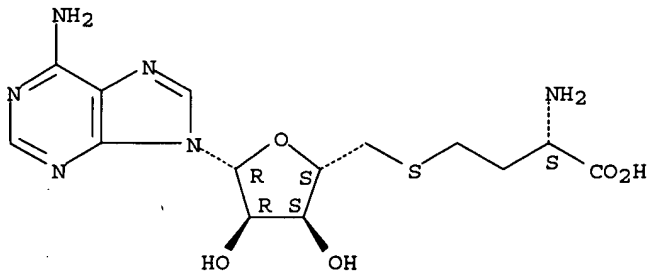


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

882 REFERENCES IN FILE CA (1907 TO DATE)
16 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
882 REFERENCES IN FILE CAPLUS (1907 TO DATE)
5 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L7 ANSWER 10 OF 20 REGISTRY COPYRIGHT 2005 ACS on STN
RN **979-92-0** REGISTRY
ED Entered STN: 16 Nov 1984
CN L-Homocysteine, S-(5'-deoxyadenosin-5'-yl)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Adenosine, 5'-S-(3-amino-3-carboxypropyl)-5'-thio-, L- (8CI)
CN Homocysteine, S-adenosyl-, L- (6CI, 7CI)
OTHER NAMES:
CN Adenosyl-L-homocysteine
CN Adenosylhomocysteine
CN L-S-Adenosylhomocysteine
CN S-Adenosyl-L-homocysteine
CN S-Adenosylhomocysteine
FS STEREOSEARCH
DR 120220-04-4, 15519-61-6, 21593-56-6, 22620-01-5, 361436-44-4
MF C14 H20 N6 O5 S
CI COM
LC STN Files: AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CEN, CHEMCATS, CHEMLIST, CSCHEM, DDFU, DRUGU, EMBASE, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, PROMT, SPECINFO, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.

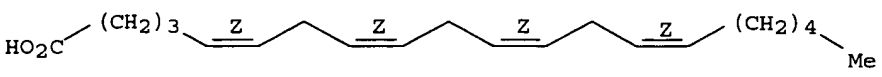


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

1213 REFERENCES IN FILE CA (1907 TO DATE)
108 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
1214 REFERENCES IN FILE CAPLUS (1907 TO DATE)
16 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L7 ANSWER 11 OF 20 REGISTRY COPYRIGHT 2005 ACS on STN
RN **506-32-1** REGISTRY
ED Entered STN: 16 Nov 1984
CN 5,8,11,14-Eicosatetraenoic acid, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 5,8,11,14-Eicosatetraenoic acid, (all-Z)- (8CI)
OTHER NAMES:
CN (all-Z)-5,8,11,14-Eicosatetraenoic acid
CN 5,8,11,14-all-cis-Eicosatetraenoic acid
CN 5-cis,8-cis,11-cis,14-cis-Eicosatetraenoic acid
CN 5Z,8Z,11Z,14Z-Eicosatetraenoic acid
CN all-cis-5,8,11,14-Eicosatetraenoic acid
CN arachidonate
CN Arachidonic acid
CN cis-Δ5,8,11,14-Eicosatetraenoic acid
FS STEREOSEARCH
DR 10417-93-3, 929-92-0
MF C20 H32 O2
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DIOGENES, DRUGU, EMBASE, GMELIN*, HODOC*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, NAPRALERT, NIOSHTIC, PATDPASPC, PROMT, RTECS*, SPECINFO, TOXCENTER, USPAT2, USPATFULL, VETU, VTB
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

Double bond geometry as shown.



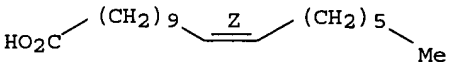
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

28996 REFERENCES IN FILE CA (1907 TO DATE)
2280 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
29019 REFERENCES IN FILE CAPLUS (1907 TO DATE)

132 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L7 ANSWER 12 OF 20 REGISTRY COPYRIGHT 2005 ACS on STN
RN 506-17-2 REGISTRY
ED Entered STN: 16 Nov 1984
CN 11-Octadecenoic acid, (11Z)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 11-Octadecenoic acid, (Z)- (8CI)
CN 11-Octadecenoic acid, cis- (6CI)
OTHER NAMES:
CN (Z)-11-Octadecenoic acid
CN Δ11-cis-Octadecenoic acid
CN 11(Z)-Octadecenoic acid
CN 11-cis-Octadecenoic acid
CN 11-cis-Vaccenic acid
CN 11Z-Octadecenoic acid
CN Asclepic acid
CN cis-Δ11-Octadecenoic acid
CN cis-11-Octadecenoic acid
CN cis-11-Vaccenic acid
CN cis-Vaccenic acid
CN cis-Vaccenic acid
FS STEREOSEARCH
MF C18 H34 O2
CI COM
LC STN Files: AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CHEMCATS, CSCHEM, DETHERM*, IPA, MEDLINE, NIOSHTIC, PROMT, SPECINFO, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)

Double bond geometry as shown.



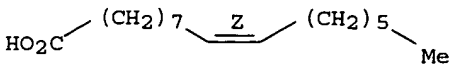
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

2687 REFERENCES IN FILE CA (1907 TO DATE)
20 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
2694 REFERENCES IN FILE CAPLUS (1907 TO DATE)
10 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L7 ANSWER 13 OF 20 REGISTRY COPYRIGHT 2005 ACS on STN
RN 373-49-9 REGISTRY
ED Entered STN: 16 Nov 1984
CN 9-Hexadecenoic acid, (9Z)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 9-Hexadecenoic acid, (Z)- (8CI)
CN Palmitoleic acid (7CI)
OTHER NAMES:
CN (Z)-9-Hexadecenoic acid
CN 9-cis-Hexadecenoic acid
CN 9-Hexadecenoic acid
CN 9Z-Hexadecenoic acid
CN 9Z-Hexadecenoic acid
CN cis-Δ9-Hexadecenoic acid
CN cis-9-Hexadecenoic acid
CN cis-Palmitoleic acid
CN Oleopalmitic acid
CN Zoomeric acid
FS STEREOSEARCH

MF C16 H30 O2
CI COM
LC STN Files: AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CAPLUS, CASREACT, CHEMCATS, CHEMLIST, CIN, CSCHEM, DDFU, DRUGU, EMBASE, HODOC*, IFICDB, IFIPAT, IFIUDB, IPA, NAPRALERT, NIOSHTIC, PATDPASPC, PIRA, PROMT, SPECINFO, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

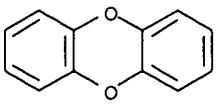
Double bond geometry as shown.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

9109 REFERENCES IN FILE CA (1907 TO DATE)
148 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
9129 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L7 ANSWER 14 OF 20 REGISTRY COPYRIGHT 2005 ACS on STN
RN **262-12-4** REGISTRY
ED Entered STN: 16 Nov 1984
CN Dibenzo[b,e][1,4]dioxin (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Dibenzo-p-dioxin (6CI, 7CI, 8CI)
OTHER NAMES:
CN Dibenzo[1,4]dioxin
CN Diphenylene dioxide
CN Oxanthrene
CN Phenodioxin
FS 3D CONCORD
DR 343946-29-2
MF C12 H8 O2
CI COM, RPS
LC STN Files: AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, DETHERM*, EMBASE, GMELIN*, HODOC*, HSDB*, IFICDB, IFIPAT, IFIUDB, MEDLINE, NIOSHTIC, PIRA, PROMT, RTECS*, SPECINFO, TOXCENTER, ULIDAT, USPAT2, USPATFULL, VTB
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)



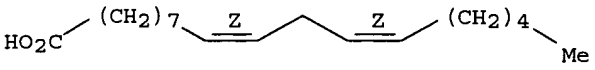
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

13257 REFERENCES IN FILE CA (1907 TO DATE)
12408 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
13264 REFERENCES IN FILE CAPLUS (1907 TO DATE)
21 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L7 ANSWER 15 OF 20 REGISTRY COPYRIGHT 2005 ACS on STN

RN 60-33-3 REGISTRY
ED Entered STN: 16 Nov 1984
CN 9,12-Octadecadienoic acid (9Z,12Z)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 9,12-Octadecadienoic acid (Z,Z)-
CN Linoleic acid (8CI)
OTHER NAMES:
CN (9Z,12Z)-9,12-Octadecadienoic acid
CN (Z,Z)-9,12-Octadecadienoic acid
CN α-Linoleic acid
CN 9,12-Octadecadienoic acid, (Z,Z)-
CN 9-cis,12-cis-Linoleic acid
CN 9Z,12Z-Linoleic acid
CN 9Z,12Z-Octadecadienoic acid
CN all-cis-9,12-Octadecadienoic acid
CN cis,cis-Linoleic acid
CN cis-Δ9,12-Octadecadienoic acid
CN cis-9,cis-12-Octadecadienoic acid
CN Emersol 315
CN Extra Linoleic 90
CN Linolic acid
CN Polylin 515
CN Unifac 6550
FS STEREOSEARCH
MF C18 H32 O2
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DIOGENES, DIPPR*, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT; ENCOMPPAT2, GMELIN*, HODOC*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PATDPASPC, PDLCOM*, PIRA, PROMT, RTECS*, SPECINFO, TOXCENTER, TULSA, USPAT2, USPATFULL, VETU
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

Double bond geometry as shown.

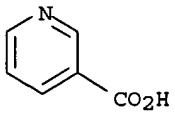


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

33431 REFERENCES IN FILE CA (1907 TO DATE)
1393 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
33476 REFERENCES IN FILE CAPLUS (1907 TO DATE)
10 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L7 ANSWER 16 OF 20 REGISTRY COPYRIGHT 2005 ACS on STN
RN 59-67-6 REGISTRY
ED Entered STN: 16 Nov 1984
CN 3-Pyridinecarboxylic acid (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Nicotinic acid (7CI, 8CI)
OTHER NAMES:
CN β-Pyridinecarboxylic acid
CN 3-Carboxylpyridine
CN 3-Carboxypyridine
CN 3-Pyridylcarboxylic acid
CN Akotin
CN Apelagrin
CN Daskil

CN E 375
CN Efacin
CN Enduracin
CN Linic
CN Niac
CN Niacin
CN Niacor
CN Niaspan
CN Nicacid
CN Nicangin
CN Nico-Span
CN Nicobid
CN Nicodelmine
CN Nicolar
CN Niconacid
CN Nicosan 3
CN Nicotinipca
CN Nicyl
CN NSC 169454
CN Nyclin
CN Pellagrin
CN Pelonin
CN Slo-niacin
CN SR 4390
CN Vitamin B5
CN Wampocap
FS 3D CONCORD
DR 123574-58-3
MF C6 H5 N O2
CI COM
LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DIOGENES, DIPPR*, DRUGU, EMBASE, GMELIN*, HODOC*, HSDB*, IFICDB, IFIPAT, IFIUDB, IMSCOSEARCH, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PDLCOM*, PHAR, PIRA, PROMT, PROUSDDR, PS, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, TULSA, ULIDAT, USAN, USPAT2, USPATFULL, VETU, VTB
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)



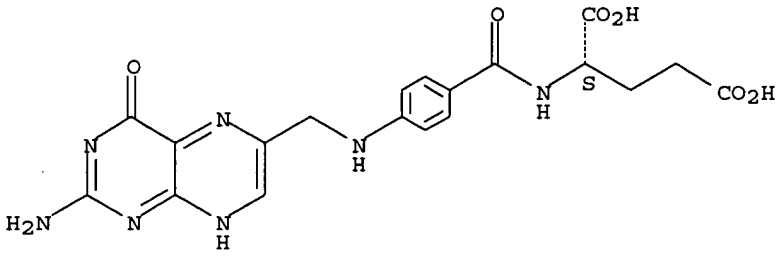
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

15519 REFERENCES IN FILE CA (1907 TO DATE)
710 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
15534 REFERENCES IN FILE CAPLUS (1907 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L7 ANSWER 17 OF 20 REGISTRY COPYRIGHT 2005 ACS on STN
RN 59-30-3 REGISTRY
ED Entered STN: 16 Nov 1984
CN L-Glutamic acid, N-[4-[[(2-amino-1,4-dihydro-4-oxo-6-pteridinyl)methyl]amino]benzoyl]- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Folic acid (8CI)
OTHER NAMES:

CN Acifolic
CN Cytofol
CN Dosfolat B activ
CN Folacid
CN Folacin
CN Folbal
CN Folcidin
CN Foldine
CN Folettes
CN Foliamin
CN Folicet
CN Folipac
CN Folsan
CN Folsaure
CN Folsav
CN Folvite
CN Incafolic
CN Liver Lactobacillus casei factor
CN Millafol
CN NSC 3073
CN PGA
CN Pteroyl-L-glutamic acid
CN Pteroyl-L-monoglutamic acid
CN Pteroylglutamic acid
CN Pteroylmonoglutamic acid
CN Vitamin Bc
CN Vitamin Be
CN Vitamin M
FS STEREOSEARCH
DR 33609-88-0
MF C19 H19 N7 O6
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DIOGENES, DRUGU, EMBASE, GMELIN*, HODOC*, HSDB*, IFICDB, IFIPAT, IFIUDB, IMSCOSEARCH, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PIRA, PROMT, PS, RTECS*, SPECINFO, TOXCENTER, USAN, USPAT2, USPATFULL, VETU
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



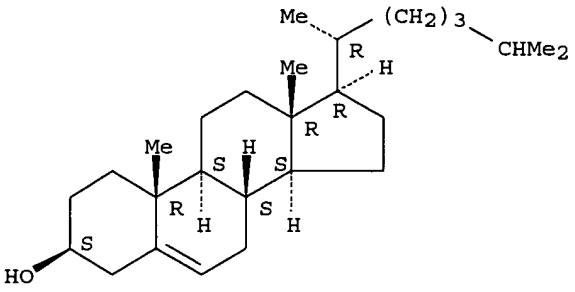
PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

15099 REFERENCES IN FILE CA (1907 TO DATE)
1087 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
15114 REFERENCES IN FILE CAPLUS (1907 TO DATE)
9 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L7 ANSWER 18 OF 20 REGISTRY COPYRIGHT 2005 ACS on STN
RN 57-88-5 REGISTRY

ED Entered STN: 16 Nov 1984
CN Cholest-5-en-3-ol (3β)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Cholesterol (8CI)
OTHER NAMES:
CN (-)-Cholesterol
CN Δ5-Cholesten-3β-ol
CN 3β-Hydroxycholest-5-ene
CN 5:6-Cholesten-3β-ol
CN Cholest-5-en-3β-ol
CN Cholesterin
CN Cholesteryl alcohol
CN Dythol
CN Lidinit
CN Lidinite
CN NSC 8798
CN Provitamin D
FS STEREOSEARCH
DR 849593-11-9, 732297-95-9, 793670-51-6, 80356-14-5, 80356-33-8, 209124-38-9, 218965-24-3, 262418-13-3, 378185-03-6, 676322-57-9
MF C27 H46 O
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DIOGENES, DIPPR*, DRUGU, EMBASE, GMELIN*, HODOC*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PDLCOM*, PIRA, PROMT, RTECS*, SPECINFO, TOXCENTER, TULSA, ULIDAT, USAN, USPAT2, USPATFULL, VETU, VTB
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

109100 REFERENCES IN FILE CA (1907 TO DATE)
9641 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
109177 REFERENCES IN FILE CAPLUS (1907 TO DATE)
15 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L7 ANSWER 19 OF 20 REGISTRY COPYRIGHT 2005 ACS on STN
RN 57-11-4 REGISTRY
ED Entered STN: 16 Nov 1984
CN Octadecanoic acid (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1-Heptadecanecarboxylic acid
CN 17FA
CN 400JB9103-88
CN A 1760
CN Adeka Fatty Acid SA 910

CN Barolub FTA
CN Century 1210
CN Century 1220
CN Century 1230
CN Century 1240
CN Edenor C 18/98
CN Edenor C18
CN Edenor HT-JG 60
CN Edenor ST 1
CN Edenor ST 20
CN Emersol 120
CN Emersol 153NF
CN Emersol 6349
CN F 3
CN F 3 (lubricant)
CN FA 1655
CN G 270
CN Humko Industrene R
CN Hydrofol Acid 150
CN Hydrofol Acid 1895
CN Hystrene 5016
CN Hystrene 80
CN Hystrene 9718
CN Hystrene 9718NF
CN Hystrene 9718NFFG
CN Hystrene S 97
CN Hystrene T 70
CN Industrene 5016K
CN Industrene 8718
CN Industrene 9018
CN Industrene R
CN Kam 1000
CN Kam 2000
CN Kam 3000
CN Kortacid 1895
CN Loxiol G 20
CN Lunac 30
CN Lunac S 20
CN Lunac S 30
CN Lunac S 40
CN Lunac S 50
CN Lunac S 90
CN Lunac S 90KC
CN Lunac S 98
CN Lunac YA
ADDITIONAL NAMES NOT AVAILABLE IN THIS FORMAT - Use FCN, FIDE, or ALL for DISPLAY
FS 3D CONCORD
DR 8013-28-3, 8023-06-1, 8037-40-9, 8037-83-0, 8039-51-8, 8039-52-9, 8039-53-0, 8039-54-1, 58392-66-8, 134503-33-6, 82497-27-6, 39390-61-9, 197923-10-7, 294203-07-9
MF C18 H36 O2
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CHEMSAFE, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DIOGENES, DIPPR*, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT, ENCOMPPAT2, GMELIN*, HODOC*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PATDPASPC, PDLCOM*, PIRA, PROMT, PS, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, TULSA, USAN, USPAT2, USPATFULL, VETU, VTB
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

Search done by Noble Jarrell

$HO_2C-(CH_2)_{16}-Me$

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

46455 REFERENCES IN FILE CA (1907 TO DATE)
3448 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
46520 REFERENCES IN FILE CAPLUS (1907 TO DATE)
19 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L7 ANSWER 20 OF 20 REGISTRY COPYRIGHT 2005 ACS on STN
RN 57-10-3 REGISTRY
ED Entered STN: 16 Nov 1984
CN Hexadecanoic acid (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Palmitic acid (7CI, 8CI)
OTHER NAMES:
CN 1-Pentadecanecarboxylic acid
CN Cetylic acid
CN Edenor C16
CN Emersol 143
CN FA 1695
CN Hydrofol Acid 1690
CN Hystrene 9016
CN Kortacid 1698
CN Loxiol EP 278
CN Lunac P 95
CN Lunac P 95KC
CN Lunac P 98
CN n-Hexadecanoic acid
CN n-Hexadecoic acid
CN NAA 160
CN Neo-Fat 16
CN NSC 5030
CN PA 900
CN Palmitinic acid
CN Pentadecanecarboxylic acid
CN Prifac 2960
CN Pristerene 4934
FS 3D CONCORD
DR 60605-23-4, 66321-94-6, 116860-99-2, 212625-86-0
MF C16 H32 O2
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DIPPR*, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT, ENCOMPPAT2, GMELIN*, HODOC*, HSDB*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NAPRALERT, NIOSHTIC, PATDPASPC, PDLCOM*, PIRA, PROMT, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, TULSA, ULIDAT, USAN, USPAT2, USPATFULL, VETU, VTB
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**
(**Enter CHEMLIST File for up-to-date regulatory information)

$HO_2C-(CH_2)_{14}-Me$

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

38879 REFERENCES IN FILE CA (1907 TO DATE)
1506 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
38940 REFERENCES IN FILE CAPLUS (1907 TO DATE)

1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

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FILE 'WPIX' ENTERED AT 07:55:37 ON 10 JUN 2005
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FILE LAST UPDATED:          8 JUN 2005      <20050608/UP>
MOST RECENT DERWENT UPDATE:     200536        <200536/DW>
DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE

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http://thomsonderwent.com/support/dwpiref/reftools/classification/code-revision/
    FOR DETAILS. <<<

=> d iall l8 tot


L8   ANSWER 1 OF 1  WPIX  COPYRIGHT 2005 THE THOMSON CORP on STN
ACCESSION NUMBER:     2003-812476 [76]   WPIX
DOC. NO. CPI:         C2003-225898
TITLE:                Regulating the level of a fatty acid in a system, useful
                      for treating cardiovascular, neurological or
                      developmental disorders, comprises regulating the
                      phosphatidylethanolamine-N-methyltransferase (PEMT)
                      activity in the system.
DERWENT CLASS:        B04 D16
INVENTOR(S):          BAILLIE, R A; WATKINS, S M
PATENT ASSIGNEE(S):   (BAIL-I) BAILLIE R A; (WATK-I) WATKINS S M; (LIPO-N)
                      LIPOMICS TECHNOLOGIES INC
COUNTRY COUNT:        103
PATENT INFORMATION:

     PATENT NO      KIND DATE     WEEK    LA   PG MAIN IPC
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            LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
         W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
            DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR
            KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT
            RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA
            ZM ZW
     US 2004024065   A1 20040205 (200411)          A61K031-202
     AU 2003225726   A1 20030929 (200432)          C12N000-00
     EP 1490076      A2 20041229 (200502)  EN      A61K035-12
         R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LI LT LU LV
            MC MK NL PT RO SE SI SK TR
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APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE	
WO 2003078574	A2		WO 2003-US7242	20030307	
US 2004024065	A1	Provisional	US 2002-363587P	20020311	<--
		Provisional	US 2002-373912P	20020419	<--
		Provisional	US 2002-401684P	20020806	<--
		Provisional	US 2002-424949P	20021108	<--
		Provisional	US 2002-436192P	20021224	<--
			US 2003-383850	20030307	
AU 2003225726	A1		AU 2003-225726	20030307	
EP 1490076	A2		EP 2003-744631	20030307	
			WO 2003-US7242	20030307	

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2003225726	A1	Based on	WO 2003078574
EP 1490076	A2	Based on	WO 2003078574

PRIORITY APPLN. INFO: **US 2002-436192P 20021224; US 2002-363587P 20020311; US 2002-373912P 20020419; US 2002-401684P 20020806; US 2002-424949P 20021108**; US 2003-383850 20030307

INT. PATENT CLASSIF.:

MAIN: A61K031-202; A61K035-12; C12N000-00

SECONDARY: A61K031-07; A61K031-20; A61K031-685

BASIC ABSTRACT:

WO2003078574 A UPAB: 20031125

NOVELTY - Regulating the level of a fatty acid in a system comprising regulating the phosphatidylethanolamine-N-methyltransferase (PEMT) activity in the system to regulate the level of a fatty acid in the system, where the fatty acid is selected from 16:0, 18:0, 16:1n7, 18:1n7, 18:2n6, 20:4n6, 20:5n3, 22:6n3, saturated fatty acids, polyunsaturated fatty acids, and highly unsaturated fatty acids, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) decreasing the level of a fatty acid in a system;

(2) increasing the PEMT activity in a subject;

(3) regulating a lipoprotein component ratio in a system;

(4) regulating the density or size of a lipoprotein in a system;

(5) assessing the density or size of a lipoprotein in a system;

(6) treating or preventing a cardiovascular or neurological condition, or a condition associated with PEMT activity;

(7) assessing the susceptibility of a subject to a cardiovascular or developmental condition;

(8) assessing the susceptibility of a subject to the toxicity of an agent;

(9) assessing the susceptibility of a subject to a condition associated with infertility;

(10) assessing the exposure of a subject to an agent in an environment;

(11) assessing a condition in a subject;

(12) predicting effectiveness of an agent for treating neoplasia;

(13) increasing milk production in a subject;

(14) increasing the lipid or phospholipid content of milk in a subject; and

(15) preventing a birth defect of a subject.

ACTIVITY - Cardiant; Antiarteriosclerotic; Antilipemic; Nootropic; Neuroprotective; Dermatological; Antiinflammatory; Cytostatic; Hepatotropic; Anorectic; Immunosuppressive; Antirheumatic; Antiarthritic. No biological data given.

MECHANISM OF ACTION - Gene therapy.

USE - The methods and agents are useful for preventing or treating a cardiovascular condition, e.g. atherosclerosis, hyperlipidemia, hypercholesterolemia, hypertriglyceridemia, hypertension, hyperglycemia, stroke, ischemic heart diseases, cardiomyopathy, arrhythmia, coronary artery diseases, and heart attack; a neurological condition, e.g. neurodevelopment or neurodegeneration of the subject, Asperger's Syndrome, Attention Deficit Hyperactivity Disorder, Autism spectrum disorder, Cerebral Palsy, Dysthymic Disorder, Fragile X Syndrome, Perinatally Acquired HIV Disease, Tourette's Syndrome, Alternating Hemiplegia of Childhood, Congenital Anomalies, Amold-Chiari Malformation, Meningocele, Spina Bifida, Dandy Walker Syndrome, vascular malformations, Holoprosencephaly, and Hydranencephaly, Alpers' disease, Alzheimer's Disease, Autosomal Dominant Neurodegenerative Disorder, Batten Disease, Cerebral calcinosis, Cockayne Syndrome, corticobasal, ganglionic degeneration, Dementia with Lewy Bodies, Lewy Body Variant, Alzheimer's Disease, Motor Neuron Disease, Multiple System Atrophy, Parkinson Plus syndrome, Neuronal intranuclear inclusion disease, Olivopontocerebellar Atrophy, Parkinsonian Syndromes, Pick's disease, Postpoliomyelitis Syndrome, Progressive Supranuclear Palsy, Rett Syndrome, Shy-Drager Syndrome, Tauopathies, Tri-nucleotide repeat diseases, and Tuberous Sclerosis; a developmental disorder, e.g. birth defects, congenital developmental conditions, Microcephaly and mental retardation of uncontrolled phenytketonuria (PKU); or a condition associated with the PEMT activity, e.g. cirrhosis, skin disorders, neoplasia, infectious diseases, inflammatory diseases, bone density, eicosanoid production, osteoporosis, insulin sensitivity or resistance, diabetes, obesity, renal function loss, autoimmune diseases, and atopic diseases, lipid accumulation in liver, liver steatosis, complications associated with liver transplantation, growth hormone deficiency or overproduction, thyroid hormone disorders, skin disorders, infectious diseases, rheumatoid arthritis, osteoporosis, atopic diseases, cirrhosis, renal functional loss, autoimmune diseases, lipid accumulation in liver, complications associated with liver transplantation, insulin resistance, growth hormone deficiency or excess, insulin resistance, neoplasia, or chronic bowel inflammation (all claimed).

Dwg.0/20

FILE SEGMENT: CPI
FIELD AVAILABILITY: AB
MANUAL CODES: CPI: B11-C08E; B12-K04A; B12-K04E; B14-C03; B14-E12; B14-F01A; B14-F01B; B14-F02B; B14-F02D; B14-F07; B14-F09; B14-G02D; B14-H01; B14-J01A3; B14-J01A4; B14-J01B3; B14-N01; B14-N10; B14-N11; B14-N12; B14-N16; B14-N17; B14-S04; D05-H09

=> b home
FILE 'HOME' ENTERED AT 07:55:42 ON 10 JUN 2005

=>

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=> d his full

     (FILE 'HOME' ENTERED AT 07:50:20 ON 10 JUN 2005)

     FILE 'HCAPLUS' ENTERED AT 07:50:59 ON 10 JUN 2005
                E WATKINS S/AU
L1           26 SEA ABB=ON  PLU=ON  ("WATKINS S"/AU OR "WATKINS S M"/AU)
                E WATKINS STEVEN/AU
L2           24 SEA ABB=ON  PLU=ON  ("WATKINS STEVEN"/AU OR "WATKINS STEVEN
                M"/AU OR "WATKINS STEVEN MICHAEL"/AU)
                E WATKINS STEPHEN/AU
L3           10 SEA ABB=ON  PLU=ON  ("WATKINS STEPHEN"/AU OR "WATKINS STEVE"/AU
                )
L4           12 SEA ABB=ON  PLU=ON  LIPOMICS/CS,PA
L5            1 SEA ABB=ON  PLU=ON  (US2002-436192# OR US2002-424949# OR
                US2002-401684# OR US2002-373912# OR US2002-363587#)/AP,PRN

     FILE 'REGISTRY' ENTERED AT 07:54:30 ON 10 JUN 2005

     FILE 'HCAPLUS' ENTERED AT 07:54:32 ON 10 JUN 2005
L6              TRA L5 1- RN :       20 TERMS

     FILE 'REGISTRY' ENTERED AT 07:54:32 ON 10 JUN 2005
L7           20 SEA ABB=ON  PLU=ON  L6

     FILE 'WPIX' ENTERED AT 07:54:36 ON 10 JUN 2005
L8            1 SEA ABB=ON  PLU=ON  (US2002-436192# OR US2002-424949# OR
                US2002-401684# OR US2002-373912# OR US2002-363587#)/AP,PRN

     FILE 'MEDLINE' ENTERED AT 08:18:11 ON 10 JUN 2005
                E WATKINS S/AU
L9          181 SEA ABB=ON  PLU=ON  ("WATKINS S"/AU OR "WATKINS S M"/AU)
                E WATKINS STEVE/AU
L10          11 SEA ABB=ON  PLU=ON  ("WATKINS STEVE"/AU OR "WATKINS STEVEN
                M"/AU)
L11           7 SEA ABB=ON  PLU=ON  LIPOMICS/CS
L12          24 SEA ABB=ON  PLU=ON  (L9 OR L10 OR L11) AND LIPIDS+NT/CT
L13      227698 SEA ABB=ON  PLU=ON  LIPIDS+NT/CT (L) (BI OR ME)/CT
L14          11 SEA ABB=ON  PLU=ON  L13 AND L12
L15       46750 SEA ABB=ON  PLU=ON  LIPIDS+NT/CT (L)BI/CT
L16           1 SEA ABB=ON  PLU=ON  L15 AND (L9 OR L10 OR L11)
L17      227687 SEA ABB=ON  PLU=ON  L13 NOT L14
L18        6024 SEA ABB=ON  PLU=ON  L17 AND (BIOLOGICAL MARKERS+NT OR GENETIC
                MARKERS+NT)/CT
L19       25741 SEA ABB=ON  PLU=ON  L15/MAJ
L20      302326 SEA ABB=ON  PLU=ON  (BIOLOGICAL MARKERS+NT OR GENETIC MARKERS+N
                T)/CT
L21      124975 SEA ABB=ON  PLU=ON  L20/MAJ
L22         203 SEA ABB=ON  PLU=ON  L19 AND L21
L23         192 SEA ABB=ON  PLU=ON  L22 AND PY<=2003
L24       60952 SEA ABB=ON  PLU=ON  L21 (L) (AN OR CH OR ME)/CT
L25          61 SEA ABB=ON  PLU=ON  L23 AND L24
L26             QUE ABB=ON  PLU=ON  (DIAGNOSTIC TECHNIQUES, RADIOISOTOPE+NT OR
                ISOTOPE LABELING +NT OR ISOTOPES+NT)/CT
L27       22542 SEA ABB=ON  PLU=ON  L17 AND L26
L28      430472 SEA ABB=ON  PLU=ON  (DIAGNOSTIC TECHNIQUES, RADIOISOTOPE+NT OR
                ISOTOPE LABELING +NT OR ISOTOPES+NT)/CT
L29      116390 SEA ABB=ON  PLU=ON  L28/MAJ
L30         501 SEA ABB=ON  PLU=ON  L29 AND L13/MAJ
L31         463 SEA ABB=ON  PLU=ON  L30 AND PY<=2002
L32         182 SEA ABB=ON  PLU=ON  L22 AND PY<=2002
L33          59 SEA ABB=ON  PLU=ON  L32 AND L24
                SEL AN 4 10 18 29 38-39 44-45 55 57-59 L33
L34          12 SEA ABB=ON  PLU=ON  (2000125582/AN OR 2001439982/AN OR
                2002196758/AN OR 83213694/AN OR 86282150/AN OR 87274197/AN OR
                88253285/AN OR 94294385/AN OR 95180311/AN OR 96149161/AN OR
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                96185017/AN OR 97450953/AN) AND L33
                SEL AN 8 L34
L35           1 SEA ABB=ON  PLU=ON  94294385/AN AND L34
L36         184 SEA ABB=ON  PLU=ON  ISOTOPE LABELING +NT/CT AND L15
L37       10821 SEA ABB=ON  PLU=ON  ISOTOPE LABELING +NT/CT
L38        2114 SEA ABB=ON  PLU=ON  L37/MAJ
L39           7 SEA ABB=ON  PLU=ON  L38 AND L15
L40           6 SEA ABB=ON  PLU=ON  L39 AND PY<=2002
L41       28806 SEA ABB=ON  PLU=ON  (CYTIDINE DIPHOSPHATE CHOLINE+NT OR
                PHOSPHOLIPASES+NT OR STEROL O-ACYLTRANSFERASE OR GLYCEROL-3-PHO
                SPHATE O-ACYLTRANSFERASE+NT)/CT
L42       15879 SEA ABB=ON  PLU=ON  L41/MAJ
L43        5606 SEA ABB=ON  PLU=ON  L42 AND L13
L44      122676 SEA ABB=ON  PLU=ON  L13/MAJ
L45        2815 SEA ABB=ON  PLU=ON  L44 AND L42
L46          60 SEA ABB=ON  PLU=ON  L45 AND (L20 OR L37)
L47          54 SEA ABB=ON  PLU=ON  L46 AND PY<=2002
L48           9 SEA ABB=ON  PLU=ON  L19 AND L42 AND (L20 OR L37)
L49          59 SEA ABB=ON  PLU=ON  L29 AND L19
L50           1 SEA ABB=ON  PLU=ON  L49 AND (L41 OR L42)
L51          56 SEA ABB=ON  PLU=ON  L49 AND PY<=2002
                SEL AN 46-56 37-42 34-36 29-30 24-25 27 17-21 15 5-12 2 L51
L52          40 SEA ABB=ON  PLU=ON  (1998272311/AN OR 1998382434/AN OR
                2000029087/AN OR 2000029095/AN OR 2000481149/AN OR 66071506/AN
                OR 66157647/AN OR 67003285/AN OR 67004390/AN OR 67061525/AN OR
                67126401/AN OR 67160888/AN OR 67179526/AN OR 67202029/AN OR
                67216680/AN OR 68157948/AN OR 71090573/AN OR 71288112/AN OR
                72098476/AN OR 73145444/AN OR 74062356/AN OR 77109534/AN OR
                77164650/AN OR 77176576/AN OR 77215888/AN OR 81175008/AN OR
                83100402/AN OR 85258111/AN OR 86104369/AN OR 87148087/AN OR
                90234177/AN OR 90345294/AN OR 91182817/AN OR 92044100/AN OR
                93035907/AN OR 93152551/AN OR 94358640/AN OR 96292203/AN OR
                97091885/AN OR 97161767/AN) AND L51
L53          43 SEA ABB=ON  PLU=ON  L52 OR L40 OR L35
L54           0 SEA ABB=ON  PLU=ON  L53 AND (L9 OR L10 OR L11)
                SEL AN L53 26-43 20-24 11-13 15 18  1-9
L55          37 SEA ABB=ON  PLU=ON  (1998272311/AN OR 1998382434/AN OR
                2000029087/AN OR 2000029095/AN OR 2000481149/AN OR 66071506/AN
                OR 66157647/AN OR 67003285/AN OR 67004390/AN OR 67061525/AN OR
                67126401/AN OR 67160888/AN OR 67179526/AN OR 67202029/AN OR
                67216680/AN OR 68157948/AN OR 71090573/AN OR 71288112/AN OR
                72098476/AN OR 73145444/AN OR 73216685/AN OR 74062356/AN OR
                77109534/AN OR 77176576/AN OR 77215888/AN OR 81175008/AN OR
                83100402/AN OR 85258111/AN OR 86104369/AN OR 90345294/AN OR
                92044100/AN OR 93035907/AN OR 93152551/AN OR 94358640/AN OR
                96292203/AN OR 97091885/AN OR 97161767/AN) AND L53

=> b medl

FILE 'MEDLINE' ENTERED AT 11:15:32 ON 10 JUN 2005

 FILE LAST UPDATED: 9 JUN 2005 (20050609/UP).  FILE COVERS 1950 TO DATE.

 On December 19, 2004, the 2005 MeSH terms were loaded.

 The MEDLINE reload for 2005 is now available.  For details enter HELP
 RLOAD at an arrow promt (=>).  See also:

    http://www.nlm.nih.gov/mesh/
    http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html

 OLDMEDLINE now back to 1950.

 MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the
 MeSH 2005 vocabulary.
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This file contains CAS Registry Numbers for easy and accurate substance identification.

=> d all l14 tot

L14 ANSWER 1 OF 11 MEDLINE on STN
AN 2004128525 MEDLINE
DN PubMed ID: 14668353
TI Lipopenia and skin barrier abnormalities in DGAT2-deficient mice.
AU Stone Scot J; Myers Heather M; **Watkins Steven M**; Brown Barbara E; Feingold Kenneth R; Elias Peter M; Farese Robert V Jr
CS Gladstone Institute of Cardiovascular Disease, San Francisco, California 94141-1900, USA.
NC DK56084 (NIDDK)
SO Journal of biological chemistry, (2004 Mar 19) 279 (12) 11767-76. Electronic Publication: 2003-12-10. Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200405
ED Entered STN: 20040316
Last Updated on STN: 20040528
Entered Medline: 20040527
AB The synthesis of triglycerides is catalyzed by two known acyl-CoA:diacylglycerol acyltransferase (DGAT) enzymes. Although they catalyze the same biochemical reaction, these enzymes share no sequence homology, and their relative functions are poorly understood. Gene knockout studies in mice have revealed that DGAT1 contributes to triglyceride synthesis in tissues and plays an important role in regulating energy metabolism but is not essential for life. Here we show that DGAT2 plays a fundamental role in mammalian triglyceride synthesis and is required for survival. DGAT2-deficient (Dgat2(-/-)) mice are lipopenic and die soon after birth, apparently from profound reductions in substrates for energy metabolism and from impaired permeability barrier function in the skin. DGAT1 was unable to compensate for the absence of DGAT2, supporting the hypothesis that the two enzymes play fundamentally different roles in mammalian triglyceride metabolism.
CT Acyltransferases: GE, genetics
*Acyltransferases: PH, physiology
Amino Acid Sequence
Animals
Base Sequence
Cell Line, Tumor
DNA Primers
Homeostasis
Mice
Rats
Research Support, Non-U.S. Gov't
Research Support, U.S. Gov't, P.H.S.
*Skin Abnormalities: GE, genetics
Triglycerides: BI, biosynthesis
***Triglycerides: ME, metabolism**
CN 0 (DNA Primers); 0 (Triglycerides); EC 2.3. (Acyltransferases); EC 2.3.1.20 (diacylglycerol O-acyltransferase)

L14 ANSWER 2 OF 11 MEDLINE on STN
AN 2004100027 MEDLINE
DN PubMed ID: 14991909
TI Effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) on fatty acid availability and neural tube formation in cynomolgus macaque, Macaca fascicularis.
AU Moran F M; Hendrickx A G; Shideler S; Overstreet J W; **Watkins S M**; Lasley B L
CS Center for Health and the Environment, University of California, Davis,

California 95616, USA.
SO Birth defects research. Part B, Developmental and reproductive toxicology, (2004 Feb) 71 (1) 37-46.
Journal code: 101155115. ISSN: 1542-9733.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200409
ED Entered STN: 20040302
Last Updated on STN: 20040929
Entered Medline: 20040928
AB 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) is known to alter carbohydrate utilization and specific steps in lipid metabolism. TCDD interacts with estradiol in mobilizing specific fatty acids in chickens that may be a cause of cranial/beak malformations in this species. This study was designed to test the hypothesis that TCDD simultaneously alters critical fatty acid mobilization during early pregnancy and determine if those changes correlate to morphological defects of the developing neural tube in the nonhuman primate. Cynomolgus macaques were treated with a single dose of 4 microg/kg body weight (BW) TCDD on gestational day 15 or 20. Pregnancies were terminated by hysterectomy on gestational day 24-26 and embryos were examined to determine morphology of the developing neural tube. Maternal blood samples were used for fatty acid quantification. Embryos exhibited cellular changes, mainly increased cell death, and intercellular spaces in the neural tube, suggestive of an adverse effect on the developing nervous system. Significant decreases on fatty acid composition were found on some of the eight classes of lipids analyzed. Particularly, a decrease was observed in the n-3 (40-60%) and n-6 (47-75%) essential fatty acids in treated pregnancies compared to untreated controls. These data demonstrate the effect of TCDD in decreasing maternal levels of n-3 and n-6 fatty acids that are considered necessary for normal development in mammals. Since neural tube development is dependent, in part, on n-3 and n-6 fatty acids, it is possible that the limitation of these essential fatty acids in plasma resulted in the observed detrimental effects on early brain development.

CT Check Tags: Female
Animals
Brain: PA, pathology
Embryo: DE, drug effects
Embryo: PA, pathology
Fatty Acids: AN, analysis
***Fatty Acids: ME, metabolism**
Lipid Mobilization: DE, drug effects
Lipids: BL, blood
Lipids: CH, chemistry
Macaca fascicularis
*Neural Tube Defects: CI, chemically induced
Neural Tube Defects: PA, pathology
Pregnancy
Tetrachlorodibenzodioxin: PD, pharmacology
*Tetrachlorodibenzodioxin: TO, toxicity
RN 1746-01-6 (Tetrachlorodibenzodioxin)
CN 0 (Fatty Acids); 0 (Lipids)

L14 ANSWER 3 OF 11 MEDLINE on STN
AN 2004092462 MEDLINE
DN PubMed ID: 14982154
TI Lipomic profiling in drug discovery, development and clinical trial evaluation.
AU **Watkins Steven M**
CS **Lipomics** Technologies Inc., 2545 Boatman Avenue, West Sacramento, CA 95691, USA.. **steve.watkins@lipomics.com**
SO Current opinion in drug discovery & development, (2004 Jan) 7 (1) 112-7.
Ref: 27

Journal code: 100887519. ISSN: 1367-6733.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200406
ED Entered STN: 20040302
Last Updated on STN: 20040615
Entered Medline: 20040614
AB Dysregulation of lipid metabolism plays a crucial role in the progression of many diseases and in adverse drug response. Changes in lipid metabolism leading to disease are often not detected using biomarker analyses because these changes are defined by a subtle, long-term shift in the concentrations of many metabolites simultaneously. The application of the principals of 'omics to the study of lipids, which include the development of comprehensive, high-throughput assays and centralized reference databases, is beginning to produce an integrated and actionable knowledge of lipids and their relationship to pathology. These analytical tools and the assembled knowledge will enable the discovery, development and testing of drugs to be conducted with a deeper knowledge of their effects on lipid metabolism.
CT Animals
Chromatography, Gas
Chromatography, High Pressure Liquid
Clinical Trials
Databases, Factual
*Drug Design
Humans
***Lipids: CH, chemistry**
***Lipids: ME, metabolism**
Magnetic Resonance Spectroscopy
Pharmaceutical Preparations: AE, adverse effects
Pharmaceutical Preparations: CH, chemistry
Phospholipids: CH, chemistry
Phospholipids: ME, metabolism
Spectrum Analysis, Mass
Triglycerides: CH, chemistry
Triglycerides: ME, metabolism
CN 0 (Lipids); 0 (Pharmaceutical Preparations); 0 (Phospholipids); 0 (Triglycerides)

L14 ANSWER 4 OF 11 MEDLINE on STN
AN 2003530359 MEDLINE
DN PubMed ID: 14608048
TI Phosphatidylethanolamine-N-methyltransferase activity and dietary choline regulate liver-plasma lipid flux and essential fatty acid metabolism in mice.
AU **Watkins Steven M**; Zhu Xiaonan; Zeisel Steven H
CS **Lipomics** Technologies, Incorporated, West Sacramento, CA 95691, USA.. **steve.watkins@lipomics.com**
NC AG09525 (NIA)
DK55865 (NIDDK)
DK56350 (NIDDK)
ES10126 (NIEHS)
SO Journal of nutrition, (2003 Nov) 133 (11) 3386-91.
Journal code: 0404243. ISSN: 0022-3166.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200408
ED Entered STN: 20031111
Last Updated on STN: 20040825
Entered Medline: 20040824

AB Phosphatidylethanolamine-N-methyltransferase (PEMT) catalyzes the methylation of phosphatidylethanolamine to form phosphatidylcholine (PC) and represents one of the two major pathways for PC biosynthesis. Mice with a homozygous disruption of the PEMT gene are dependent on the 1,2-diacylglycerol cholinephosphotransferase (CDP-choline) pathway for the synthesis of PC and develop severe liver steatosis when fed a diet deficient in choline. The present study used quantitative lipid metabolite profiling to characterize lipid metabolism in PEMT-deficient mice fed diets containing varying concentrations of choline. Choline supplementation restored liver, but not plasma PC concentrations of PEMT-deficient mice to levels commensurate with control mice. Choline supplementation also restored plasma triglyceride concentrations to normal levels, but did not restore plasma cholesterol ester concentrations in the PEMT-deficient mice to those equal to control mice. PEMT-deficient mice also had substantially diminished concentrations of docosahexaenoic acid [22:6(n-3)] and arachidonic acid [20:4(n-6)] in plasma, independent of choline status. Thus, choline supplementation rescued some but not all of the phenotypes induced by the knockout. These findings indicate that PEMT activity functions beyond its recognized role as a compensatory pathway for PC biosynthesis and that, in contrast, PEMT activity is involved in many physiologic processes including the flux of lipid between liver and plasma and the delivery of essential fatty acids to blood and peripheral tissues via the liver-derived lipoproteins.

CT Animals
Cholesterol Esters: ME, metabolism
Choline: AD, administration & dosage
*Choline: PD, pharmacology
Diet
***Fatty Acids, Essential: ME, metabolism**
Lipids: BL, blood
***Lipids: ME, metabolism**
Liver: DE, drug effects
*Liver: ME, metabolism
*Methyltransferases: DF, deficiency
Methyltransferases: GE, genetics
*Methyltransferases: ME, metabolism
Mice
Mice, Knockout
Phospholipids: ME, metabolism
Research Support, U.S. Gov't, P.H.S.
Triglycerides: ME, metabolism

RN 62-49-7 (Choline)

CN 0 (Cholesterol Esters); 0 (Fatty Acids, Essential); 0 (Lipids); 0 (Phospholipids); 0 (Triglycerides); EC 2.1.1. (Methyltransferases); EC 2.1.1.17 (Phosphatidylethanolamine N-methyltransferase)

L14 ANSWER 5 OF 11 MEDLINE on STN
AN 2002642802 MEDLINE
DN PubMed ID: 12401879
TI Lipid metabolome-wide effects of the PPARgamma agonist rosiglitazone.
AU **Watkins Steven M**; Reifsnyder Peter R; Pan Huei-ju; German J Bruce; Leiter Edward H
CS **Lipomics** Technologies, Inc, 2545 Boatman Ave, West Sacramento, CA 95691, USA.. **steve.watkins@lipomics.com**
NC CA-34196 (NCI)
DK56853 (NIDDK)
SO Journal of lipid research, (2002 Nov) 43 (11) 1809-17.
Journal code: 0376606. ISSN: 0022-2275.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200309
ED Entered STN: 20021029
Last Updated on STN: 20030903
Entered Medline: 20030902

AB Successful therapy for chronic diseases must normalize a targeted aspect of metabolism without disrupting the regulation of other metabolic pathways essential for maintaining health. Use of a limited number of single molecule surrogates for disease, or biomarkers, to monitor the efficacy of a therapy may fail to predict undesirable side effects. In this study, a comprehensive metabolomic assessment of lipid metabolites was employed to determine the specific effects of the peroxisome proliferator-activated receptor gamma (PPARgamma) agonist rosiglitazone on structural lipid metabolism in a new mouse model of Type 2 diabetes. Dietary supplementation with rosiglitazone (200 mg/kg diet) suppressed Type 2 diabetes in obese (NZO x NON)F1 male mice, but chronic treatment markedly exacerbated hepatic steatosis. The metabolomic data revealed that rosiglitazone i) induced hypolipidemia (by dysregulating liver-plasma lipid exchange), ii) induced de novo fatty acid synthesis, iii) decreased the biosynthesis of lipids within the peroxisome, iv) substantially altered free fatty acid and cardiolipin metabolism in heart, and v) elicited an unusual accumulation of polyunsaturated fatty acids within adipose tissue. These observations suggest that the phenotypes induced by rosiglitazone are mediated by multiple tissue-specific metabolic variables. Because many of the effects of rosiglitazone on tissue metabolism were reflected in the plasma lipid metabolome, metabolomics has excellent potential for developing clinical assessments of metabolic response to drug therapy.

CT Check Tags: Male
Adipose Tissue: DE, drug effects
Adipose Tissue: ME, metabolism
Animals
Diabetes Mellitus, Type 2: ME, metabolism
Gene Expression: DE, drug effects
Heart: DE, drug effects
Lipids: BL, blood
***Lipids: ME, metabolism**
Liver: DE, drug effects
Liver: ME, metabolism
Mice
Mice, Obese
Myocardium: ME, metabolism
*Receptors, Cytoplasmic and Nuclear: AG, agonists
Research Support, Non-U.S. Gov't
Research Support, U.S. Gov't, P.H.S.
*Thiazoles: PD, pharmacology
*Thiazolidinediones
*Transcription Factors: AG, agonists
Weight Gain

RN 122320-73-4 (rosiglitazone)

CN 0 (Lipids); 0 (Receptors, Cytoplasmic and Nuclear); 0 (Thiazoles); 0 (Thiazolidinediones); 0 (Transcription Factors)

L14 ANSWER 6 OF 11 MEDLINE on STN

AN 2002390456 MEDLINE

DN PubMed ID: 12139307

TI Metabolomics and biochemical profiling in drug discovery and development.

AU **Watkins Steven M**; German J Bruce

CS **Lipomics** Technologies Inc, West Sacramento, CA 95691, USA..
steve.watkins@lipomics.com

SO Current opinion in molecular therapeutics, (2002 Jun) 4 (3) 224-8. Ref: 21
Journal code: 100891485. ISSN: 1464-8431.

CY England: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)

LA English

FS Priority Journals

EM 200301

ED Entered STN: 20020726

Last Updated on STN: 20030110
Entered Medline: 20030109

AB Metabolomics brings a different and complementary perspective to biomedical research and therapeutic development. Metabolites are measurable, directly responsible for health, and changeable through intervention. By definition, the concentrations of metabolites are the direct reflection of metabolism, and by measuring changes in metabolite concentrations, the full range of biochemical effects induced by a therapeutic intervention can be determined. In a clinical setting, metabolomics can be used to diagnose or predict disease, stratify patient populations by their specific metabolism, or to determine the safety or efficacy of a therapeutic intervention. Metabolomics is useful in virtually all aspects of biomedical research, and it is likely to evolve into an essential component of the drug discovery and development process.

CT Animals
*Drug Design
Gene Expression Profiling
*Genomics
Humans
Lipids: AN, analysis
Lipids: CL, classification
Lipids: ME, metabolism
Magnetic Resonance Spectroscopy
*Metabolism
Metabolism, Inborn Errors: DI, diagnosis
Metabolism, Inborn Errors: GE, genetics
Metabolism, Inborn Errors: ME, metabolism
Pharmaceutical Preparations: AN, analysis
Pharmaceutical Preparations: ME, metabolism
Pharmacogenetics

CN 0 (Lipids); 0 (Pharmaceutical Preparations)

L14 ANSWER 7 OF 11 MEDLINE on STN
AN 2002016566 MEDLINE
DN PubMed ID: 11423386
TI Interaction of estrogen and 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) with hepatic fatty acid synthesis and metabolism of male chickens (Gallus domesticus).
AU Stanton B; **Watkins S**; German J B; Lasley B
CS Institute of Toxicology and Environmental Health (ITEH), University of California Davis, One Shields Ave., 95616, Davis, CA, USA.. rjstanton@ucdavis.edu
SO Comparative biochemistry and physiology. Toxicology & pharmacology : CBP, (2001 Jun) 129 (2) 137-50.
Journal code: 100959500. ISSN: 1532-0456.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200112
ED Entered STN: 20020121
Last Updated on STN: 20020121
Entered Medline: 20011204

AB This study tested the hypothesis that 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) antagonizes estrogen-induced hepatic lipid synthesis and metabolism in birds. Twenty immature male chickens (Gallus domesticus) were divided evenly into four groups: (1) vehicle control; (2) estrogen alone (1.0 mg/kg estradiol cypionate injected on three consecutive days); (3) TCDD alone (50 microg/kg injected on the fourth day); and (4) a combination of the estrogen and TCDD treatments. On day 14, liver samples were collected for quantitative fatty acid analysis by capillary gas chromatography. Birds treated with estrogen alone had increased total triacylglyceride concentrations with specific increases in the Delta9 desaturase products 16:1n7, 18:1n7, 18:1n9, and 20:1n9. In addition, estrogen treatment specifically increased 22:6n3 concentrations in both triacylglycerides and phospholipids. However, these increases in Delta9 desaturase products or

22:6n3 did not occur for birds treated with estrogen in combination with TCDD. TCDD and estrogen plus TCDD treatments increased phospholipid concentrations of the diet-derived polyunsaturated fatty acids 18:2n6, 18:3n6, 20:3n6, 18:3n3, and 20:5n3, although only the estrogen plus TCDD group had significantly increased total phospholipids. In cholesterol esters, all three treatments decreased concentrations of total fatty acids, saturated fatty acids, and Delta9 desaturase products compared to the control group.

CT Check Tags: Male
Animals
*Chickens: ME, metabolism
Chromatography, Gas
Chromatography, Thin Layer
Dose-Response Relationship, Drug
Drug Antagonism
*Environmental Pollutants: PD, pharmacology
*Estrogen Antagonists: PD, pharmacology
***Fatty Acids: ME, metabolism**
Liver: DE, drug effects
*Liver: ME, metabolism
Phospholipids: ME, metabolism
Research Support, Non-U.S. Gov't
Research Support, U.S. Gov't, Non-P.H.S.
Tetrachlorodibenzodioxin: ME, metabolism
*Tetrachlorodibenzodioxin: PD, pharmacology
Triglycerides: ME, metabolism
Wasting Syndrome: CI, chemically induced
Wasting Syndrome: VE, veterinary

RN 1746-01-6 (Tetrachlorodibenzodioxin)

CN 0 (Environmental Pollutants); 0 (Estrogen Antagonists); 0 (Fatty Acids); 0 (Phospholipids); 0 (Triglycerides)

L14 ANSWER 8 OF 11 MEDLINE on STN

AN 2001691991 MEDLINE

DN PubMed ID: 11739435

TI Skeletal muscle lipid content and insulin resistance: evidence for a paradox in endurance-trained athletes.

AU Goodpaster B H; He J; **Watkins S**; Kelley D E

CS Department of Medicine, University of Pittsburgh, Pittsburgh, Pennsylvania 15261, USA.. bgood+@pitt.edu

NC 1P30DK46204 (NIDDK)
5M01RR00056 (NCRR)
K01 AG00851 (NIA)
R01 DK-49200-04 (NIDDK)

SO Journal of clinical endocrinology and metabolism, (2001 Dec) 86 (12) 5755-61.
Journal code: 0375362. ISSN: 0021-972X.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Abridged Index Medicus Journals; Priority Journals

EM 200112

ED Entered STN: 20011213
Last Updated on STN: 20020124
Entered Medline: 20011228

AB We examined the hypothesis that an excess accumulation of intramuscular lipid (IMCL) is associated with insulin resistance and that this may be mediated by the oxidative capacity of muscle. Nine sedentary lean (L) and 11 obese (O) subjects, 8 obese subjects with type 2 diabetes mellitus (D), and 9 lean, exercise-trained (T) subjects volunteered for this study. Insulin sensitivity (M) determined during a hyperinsulinemic (40 mU x m(-2)min(-1)) euglycemic clamp was greater ($P < 0.01$) in L and T, compared with O and D (9.45 +/- 0.59 and 10.26 +/- 0.78 vs. 5.51 +/- 0.61 and 1.15 +/- 0.83 mg x min(-1)kg fat free mass(-1), respectively). IMCL in percutaneous vastus lateralis biopsy specimens by quantitative image analysis of Oil Red O staining was approximately 2-fold higher in D than

in L (3.04 +/- 0.39 vs. 1.40 +/- 0.28% area as lipid; P < 0.01). IMCL was also higher in T (2.36 +/- 0.37), compared with L (P < 0.01). The oxidative capacity of muscle determined with succinate dehydrogenase staining of muscle fibers was higher in T, compared with L, O, and D (50.0 +/- 4.4, 36.1 +/- 4.4, 29.7 +/- 3.8, and 33.4 +/- 4.7 optical density units, respectively; P < 0.01). IMCL was negatively associated with M (r = -0.57, P < 0.05) when endurance-trained subjects were excluded from the analysis, and this association was independent of body mass index. However, the relationship between IMCL and M was not significant when trained individuals were included. There was a positive association between the oxidative capacity and M among nondiabetics (r = 0.37, P < 0.05). In summary, skeletal muscle of trained endurance athletes is markedly insulin sensitive and has a high oxidative capacity, despite having an elevated lipid content. In conclusion, the capacity for lipid oxidation may be an important mediator of the association between excess muscle lipid accumulation and insulin resistance.

CT Check Tags: Female; Male
Adult
Exercise
Humans
*Insulin Resistance
***Lipids: ME, metabolism**
Middle Aged
*Muscle, Skeletal: ME, metabolism
Oxidation-Reduction
*Physical Education and Training
*Physical Endurance
Research Support, U.S. Gov't, P.H.S.
Succinate Dehydrogenase: ME, metabolism

CN 0 (Lipids); EC 1.3.99.1 (Succinate Dehydrogenase)

L14 ANSWER 9 OF 11 MEDLINE on STN
AN 2001504816 MEDLINE
DN PubMed ID: 11337979
TI Unique phospholipid metabolism in mouse heart in response to dietary docosahexaenoic or alpha-linolenic acids.
AU **Watkins S M**; Lin T Y; Davis R M; Ching J R; DePeters E J; Halpern G M; Walzem R L; German J B
CS Department of Food Science and Technology, 1 Shields Ave., University of California at Davis, Davis, CA 95616, USA.. smwatkins@ucdavis.edu
SO Lipids, (2001 Mar) 36 (3) 247-54.
Journal code: 0060450. ISSN: 0024-4201.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200109
ED Entered STN: 20010917
Last Updated on STN: 20010917
Entered Medline: 20010913
AB Diet and fatty acid metabolism interact in yet unknown ways to modulate membrane fatty acid composition and certain cellular functions. For example, dietary precursors or metabolic products of n-3 fatty acid metabolism differ in their ability to modify specific membrane components. In the present study, the effect of dietary 22:6n-3 or its metabolic precursor, 18:3n-3, on the selective accumulation of 22:6n-3 by heart was investigated. The mass and fatty acid compositions of individual phospholipids (PL) in heart and liver were quantified in mice fed either 22:6n-3 (from crocodile oil) or 18:3n-3 (from soybean oil) for 13 wk. This study was conducted to determine if the selective accumulation of 22:6n-3 in heart was due to the incorporation of 22:6n-3 into cardiolipin (CL), a PL most prevalent in heart and known to accumulate 22:6n-3. Although heart was significantly enriched with 22:6n-3 relative to liver, the accumulation of 22:6n-3 by CL in heart could not quantitatively account for this difference. CL from heart did accumulate 22:6n-3, but only in mice fed preformed 22:6n-3. Diets rich in non-22:6n-3 fatty acids

result in a fatty acid composition of phosphatidylcholine (PC) in heart that is unusually enriched with 22:6n-3. In this study, the mass of PC in heart was positively correlated with the enrichment of 22:6n-3 into PC. The increased mass of PC was coincident with a decrease in the mass of phosphatidylethanolamine, suggesting that 22:6n-3 induced PC synthesis by increasing phosphatidylethanolamine-N-methyltransferase activity in the heart.

CT Alligators and Crocodiles
Animals
Cardiolipins: ME, metabolism
***Dietary Fats, Unsaturated: PD, pharmacology**
***Docosahexaenoic Acids: PD, pharmacology**
Fatty Acids: AN, analysis
Lipids: AN, analysis
Liver: CH, chemistry
Liver: ME, metabolism
Mice
Myocardium: CH, chemistry
*Myocardium: ME, metabolism
Phosphatidylcholines: AN, analysis
Phosphatidylethanolamines: AN, analysis
Phospholipids: AN, analysis
***Phospholipids: ME, metabolism**
Soybean Oil: PD, pharmacology
***alpha-Linolenic Acid: PD, pharmacology**

RN 25167-62-8 (Docosahexaenoic Acids); 463-40-1 (alpha-Linolenic Acid); 8001-22-7 (Soybean Oil)

CN 0 (Cardiolipins); 0 (Dietary Fats, Unsaturated); 0 (Fatty Acids); 0 (Lipids); 0 (Phosphatidylcholines); 0 (Phosphatidylethanolamines); 0 (Phospholipids)

L14 ANSWER 10 OF 11 MEDLINE on STN
AN 2001199367 MEDLINE
DN PubMed ID: 11289047
TI Skeletal muscle lipid content and oxidative enzyme activity in relation to muscle fiber type in type 2 diabetes and obesity.
AU He J; **Watkins S**; Kelley D E
CS Department of Medicine, University of Pittsburgh School of Medicine, Pennsylvania, USA.
NC 5 M01RR00056 (NCRR)
DK49200-06 (NIDDK)
P30DK462-08 (NIDDK)
SO Diabetes, (2001 Apr) 50 (4) 817-23.
Journal code: 0372763. ISSN: 0012-1797.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200104
ED Entered STN: 20010425
Last Updated on STN: 20010425
Entered Medline: 20010419
AB In obesity and type 2 diabetes, skeletal muscle has been observed to have a reduced oxidative enzyme activity, increased glycolytic activity, and increased lipid content. These metabolic characteristics are related to insulin resistance of skeletal muscle and are factors potentially related to muscle fiber type. The current study was undertaken to examine the interactions of muscle fiber type in relation to oxidative enzyme activity, glycolytic enzyme activity, and muscle lipid content in obese and type 2 diabetic subjects compared with lean healthy volunteers. The method of single-fiber analysis was used on vastus lateralis muscle obtained by percutaneous biopsy from 22 lean, 20 obese, and 20 type 2 diabetic subjects (ages 35+/-1, 42+/-2, and 52+/-2 years, respectively), with values for BMI that were similar in obese and diabetic subjects (23.7+/-0.7, 33.2+/-0.8, and 31.8+/-0.8 kg/m2, respectively). Oxidative enzyme activity followed the order of type I > type IIa > type IIb, but

within each fiber type, skeletal muscle from obese and type 2 diabetic subjects had lower oxidative enzyme activity than muscle from lean subjects ($P < 0.01$). Muscle lipid content followed a similar pattern in relation to fiber type, and within each fiber type, muscle from obese and type 2 diabetic subjects had greater lipid content ($P < 0.01$). In summary, based on single-fiber analysis, skeletal muscle in obese and type 2 diabetic subjects mani-fests disturbances of oxidative enzyme activity and increased lipid content that are independent of the effect of fiber type.

CT Check Tags: Female; Male
Adult
*Diabetes Mellitus, Type 2: ME, metabolism
Glycolysis
Humans
***Lipids: ME, metabolism**
Middle Aged
Muscle Fibers, Fast-Twitch: ME, metabolism
Muscle Fibers, Slow-Twitch: ME, metabolism
*Muscle, Skeletal: ME, metabolism
*Obesity: ME, metabolism
*Oxidoreductases: ME, metabolism
Reference Values
Research Support, U.S. Gov't, Non-P.H.S.
Research Support, U.S. Gov't, P.H.S.
Thinness

CN 0 (Lipids); EC 1. (Oxidoreductases)

L14 ANSWER 11 OF 11 MEDLINE on STN
AN 1998381817 MEDLINE
DN PubMed ID: 9717717
TI Docosahexaenoic acid accumulates in cardiolipin and enhances HT-29 cell oxidant production.
AU **Watkins S M**; Carter L C; German J B
CS Department of Food Science and Technology, University of California, Davis 95616, USA.
NC 1 U24 AI37627 (NIAID)
SO Journal of lipid research, (1998 Aug) 39 (8) 1583-8.
Journal code: 0376606. ISSN: 0022-2275.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199811
ED Entered STN: 19990106
Last Updated on STN: 19990106
Entered Medline: 19981116

AB The objective of this study was to investigate membrane fatty acids for their effects on mitochondrial function in live cells. Mitochondrial potential and oxidant production were measured in human colonic adenocarcinoma (HT-29) cells with membranes enhanced in either oleic acid, linoleic acid, arachidonic acid, eicosapentaenoic acid, or docosahexaenoic acid. Docosahexaenoic acid-enriched cells had increased mitochondrial potential and produced 5-fold more cellular oxidants than did cells enriched with any other fatty acid. Oxidant production in fatty acid-enriched HT-29 cells did not correlate with the degree of unsaturation for total membrane fatty acids. However, there was a strong correlation between the degree of fatty acid unsaturation of cardiolipin, a critical inner-mitochondrial membrane phospholipid, and oxidant production. Cardiolipin acyl composition is known to influence the activity of electron transport complexes, an effect that can increase mitochondrial oxidant production. Docosahexaenoic acid was enriched to 48 mol% of the fatty acids present in HT-29 cell cardiolipin. These results demonstrate the importance of membrane acyl composition to mitochondrial potential and oxidant production in live cells. Additionally, results suggest that docosahexaenoic acid increases cell oxidant production by accumulating in cardiolipin, where its presence alters electron transport

efficiency.
CT **Cardiolipins: CH, chemistry**
***Cardiolipins: ME, metabolism**
Cell Line
***Docosahexaenoic Acids: ME, metabolism**
Electron Transport
Fatty Acids: ME, metabolism
Fatty Acids, Unsaturated: ME, metabolism
Humans
Membrane Lipids: CH, chemistry
Membrane Lipids: ME, metabolism
Membrane Potentials
Mitochondria: ME, metabolism
*Oxidants: ME, metabolism
Phospholipids: CH, chemistry
Phospholipids: ME, metabolism
Reactive Oxygen Species: ME, metabolism
Research Support, Non-U.S. Gov't
Research Support, U.S. Gov't, P.H.S.
RN 25167-62-8 (Docosahexaenoic Acids)
CN 0 (Cardiolipins); 0 (Fatty Acids); 0 (Fatty Acids, Unsaturated); 0 (Membrane Lipids); 0 (Oxidants); 0 (Phospholipids); 0 (Reactive Oxygen Species)

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L55 ANSWER 1 OF 37 MEDLINE on STN
AN **2000481149** MEDLINE
DN PubMed ID: 10918543
TI De novo lipogenesis in adipose tissue of lean and obese women: application of deuterated water and isotope ratio mass spectrometry.
AU Guo Z K; Cella L K; Baum C; Ravussin E; Schoeller D A
CS Department of Medicine, University of Chicago, Chicago, IL 60637, USA.
SO International journal of obesity and related metabolic disorders : journal of the International Association for the Study of Obesity, **(2000 Jul)** 24 (7) 932-7.
Journal code: 9313169. ISSN: 0307-0565.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Space Life Sciences
EM 200010
ED Entered STN: 20001019
Last Updated on STN: 20011023
Entered Medline: 20001012
AB OBJECTIVE: To evaluate the feasibility of using deuterated water and isotope ratio mass spectrometry to measure de novo fatty acid synthesis in adipose tissue, and to compare this parameter in obese and lean women. SUBJECTS: Six lean and six obese premenopausal Caucasian women in the main study and three obese Pima Indians in a pilot study. MEASUREMENTS: Deuterated water was administered orally twice daily for 14 days to create stable deuterium enrichment in body water, during which series of blood samples were collected to measure body water deuterium enrichment and deuterium incorporation into plasma total Triacylglycerol (TG) fatty acids and total cholesterol. Subcutaneous fat at different sites were sampled at the beginning and the end of deuterium administration to measure deuterium incorporation into TG fatty acids. RESULTS: Fractional de novo synthesis rate of TG fatty acids in adipose tissue was 0. 014+/-0.005 and 0.014+/-0.007% in lean and obese Caucasian women, corresponding to 2+/-0.7 and 5.6+/-3.2 g (P=0.3) of fatty acids synthesized daily, respectively. Plasma TG fatty acids and cholesterol synthesis rates were comparable to those reported previously. A pilot study showed that de novo lipid synthesis in adipose tissue of obese Pima Indians was also quantitatively minor. CONCLUSION: Human adipose tissue, like the liver, does not make a major contribution to whole body lipogenesis under eucaloric conditions.

A combination of deuterated water and isotope ratio mass spectrometry is a useful research tool for studying accumulation of de novo synthesized lipids in human adipose tissue.
ST Non-programmatic
CT Check Tags: Comparative Study; Female; Male
Adipocytes: PH, physiology
*Adipose Tissue: PH, physiology
Adult
Arizona
Body Composition: PH, physiology
Cholesterol: BL, blood
Cholesterol: CH, chemistry
***Deuterium Oxide: AD, administration & dosage**
Deuterium Oxide: DU, diagnostic use
***Fatty Acids: BI, biosynthesis**
Fatty Acids: BL, blood
Fatty Acids: CH, chemistry
Feasibility Studies
Humans
Indians, North American
Obesity: EH, ethnology
*Obesity: ME, metabolism
Pilot Projects
*Spectrum Analysis, Mass: MT, methods
RN 57-88-5 (Cholesterol); 7789-20-0 (Deuterium Oxide)
CN 0 (Fatty Acids)

L55 ANSWER 2 OF 37 MEDLINE on STN
AN **2000029095** MEDLINE
DN PubMed ID: 10563355
TI Enzymatic synthesis of [14C]ceramide, [14C]glycosphingolipids, and omega-aminoceramide.
AU Ito M; Mitsutake S; Tani M; Kita K
CS Laboratory of Marine Biochemistry, Faculty of Agriculture, Kyushu University, Fukuoka, Japan.
SO Methods in enzymology, **(2000)** 311 682-9.
Journal code: 0212271. ISSN: 0076-6879.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199912
ED Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991215
CT Amidohydrolases: ME, metabolism
Carbon Radioisotopes
***Ceramides: BI, biosynthesis**
***Glycosphingolipids: BI, biosynthesis**
***Isotope Labeling**
Research Support, Non-U.S. Gov't
CN 0 (Carbon Radioisotopes); 0 (Ceramides); 0 (Glycosphingolipids); EC 3.5. (Amidohydrolases)

L55 ANSWER 3 OF 37 MEDLINE on STN
AN **2000029087** MEDLINE
DN PubMed ID: 10563347
TI Synthesis of key precursors of radiolabeled sphingolipids.
AU Bielawska A; Szulc Z; Hannun Y A
CS Department of Biochemistry and Molecular Biology, Medical University of South Carolina, Charleston 29425, USA.
SO Methods in enzymology, **(2000)** 311 518-35.
Journal code: 0212271. ISSN: 0076-6879.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English

FS Priority Journals
EM 199912
ED Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991215
CT **Carbon Radioisotopes**
*Ceramides: CH, chemistry
***Isotope Labeling**
***Sphingolipids: BI, biosynthesis**
*Sphingomyelins: CH, chemistry
*Sphingosine: CH, chemistry
Stereoisomerism
Tritium
RN 10028-17-8 (Tritium); 123-78-4 (Sphingosine)
CN 0 (Carbon Radioisotopes); 0 (Ceramides); 0 (Sphingolipids); 0 (Sphingomyelins)

L55 ANSWER 4 OF 37 MEDLINE on STN
AN **1998382434** MEDLINE
DN PubMed ID: 9714781
TI Use of deuterium oxide to measure de novo fatty acid synthesis in normal subjects consuming different dietary fatty acid composition1.
AU Konrad S D; Cook S L; Goh Y K; French M A; Clandinin M T
CS Nutrition and Metabolism Research Group, University of Alberta, Edmonton, Alberta T6G 2P5, Canada.
SO Biochimica et biophysica acta, **(1998 Jul 31)** 1393 (1) 143-52.
Journal code: 0217513. ISSN: 0006-3002.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199810
ED Entered STN: 19981020
Last Updated on STN: 19981020
Entered Medline: 19981002
AB The effect of dietary linoleic (C18:2n-6) and palmitic acids (C16:0) on rate of hepatic de novo fatty acid synthesis was assessed in normal subjects. The diet was formulated to provide combinations of high and low levels of C18:2n-6 and C16:0. After 21 days of diet treatment, plasma triacylglycerol level and incorporation of deuterium into the plasma very low density lipoprotein triacylglycerol (VLDL-TG) pool over 24 hours was measured. Plasma triacylglycerol levels were within the normal range. Increasing dietary intake of linoleic acid decreased plasma triacylglycerol level when subjects consumed a low level of dietary palmitic acid. The relative and net amount of de novo synthesized fatty acid in the plasma VLDL-TG pool was not influenced by the diet treatments. A relationship between plasma triacylglycerol level and rate of hepatic de novo fatty acid synthesis was observed.
CT Check Tags: Female; Male
Adult
***Deuterium Oxide**
*Dietary Fats: AD, administration & dosage
Dietary Fats: AN, analysis
***Fatty Acids: BI, biosynthesis**
Humans
Linoleic Acid: AD, administration & dosage
Linoleic Acid: AN, analysis
*Liver: ME, metabolism
Palmitic Acid: AD, administration & dosage
Palmitic Acid: AN, analysis
Research Support, Non-U.S. Gov't
*Triglycerides: AN, analysis
Triglycerides: BL, blood
RN 2197-37-7 (Linoleic Acid); 57-10-3 (Palmitic Acid); 7789-20-0 (Deuterium Oxide)
CN 0 (Dietary Fats); 0 (Fatty Acids); 0 (Triglycerides)

L55 ANSWER 5 OF 37 MEDLINE on STN
AN **1998272311** MEDLINE
DN PubMed ID: 9610780
TI Validation of deuterium incorporation against sterol balance for measurement of human cholesterol biosynthesis.
AU Jones P J; Ausman L M; Croll D H; Feng J Y; Schaefer E A; Lichtenstein A H
CS School of Dietetics and Human Nutrition, Faculty of Agricultural and Environmental Sciences, McGill University, Ste-Anne-de-Bellevue, Quebec, Canada.
SO Journal of lipid research, **(1998 May)** 39 (5) 1111-7.
Journal code: 0376606. ISSN: 0022-2275.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199808
ED Entered STN: 19980910
Last Updated on STN: 19980910
Entered Medline: 19980828
AB To examine the validity of the deuterium (D) incorporation technique for measurement of human cholesterol synthesis rates, D uptake from D2O into cholesterol was compared to sterol balance in 13 subjects each under three controlled diet settings. Subjects (age 62 +/- 3.6 yr, body weight 74 +/- 4.0 kg, BMI 27 +/- 1.4) consumed weight maintenance diets enriched in either corn oil, beef tallow, or stick corn oil margarine over a 5-week period. During the final week of the study period, subjects were given 1.2 g/D2O per kg body water. D enrichment was measured in plasma water and total cholesterol over 24 h. Also, during the final week, dietary intake and fecal elimination rates of cholesterol were assessed over one 6-day period to calculate sterol balance. There was no significant difference (t = 0.858, P = 0.397) between D incorporation into cholesterol (1,183 +/- 92 mg/day) and sterol balance (1,316 +/- 125 mg/day). Among diets, net cholesterol biosynthesis measured by D incorporation agreed (r = 0.745, P = 0.0001) with values derived from sterol balance. The degree of association between methods was not influenced by the wide range of fatty acid composition of the diet fat. These data demonstrate the utility of the simple, non-restrictive deuterium incorporation method as a reliable means of determining cholesterol biosynthesis in free-living humans.
CT Check Tags: Female; Male
Adult
Aged
***Cholesterol: BI, biosynthesis**
***Deuterium Oxide: PD, pharmacology**
Dietary Fats: ME, metabolism
Humans
*Indicators and Reagents: PK, pharmacokinetics
Middle Aged
Reproducibility of Results
Research Support, Non-U.S. Gov't
Research Support, U.S. Gov't, Non-P.H.S.
RN 57-88-5 (Cholesterol); 7789-20-0 (Deuterium Oxide)
CN 0 (Dietary Fats); 0 (Indicators and Reagents)

L55 ANSWER 6 OF 37 MEDLINE on STN
AN **97161767** MEDLINE
DN PubMed ID: 9008871
TI Measuring lipogenesis and cholesterol synthesis in humans with deuterated water: use of simple gas chromatographic/mass spectrometric techniques.
AU Diraison F; Pachiaudi C; Beylot M
CS Laboratoire de Physiologie Metabolique et Renale, Faculte de Medecine R. Laennec, Lyon, France.
SO Journal of mass spectrometry : JMS, **(1997 Jan)** 32 (1) 81-6.
Journal code: 9504818. ISSN: 1076-5174.
CY ENGLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199702
ED Entered STN: 19970305
Last Updated on STN: 19970305
Entered Medline: 19970219
AB Lipogenesis and cholesterol synthesis can be studied by measuring the incorporation into fatty acids and cholesterol of deuterium from deuterated water. This has been previously achieved in human subjects using low levels of deuterium enrichment in plasma water, and thus in fatty acids and cholesterol. For the measurement of enrichment in lipids, this required the use of isotope ratio mass spectrometry, a tedious and time-consuming technique. It is shown that these measurements can be performed using the much simpler gas chromatography/mass spectrometry if higher, but always safe, deuterium enrichments in plasma water are obtained. Normal subjects ingested deuterated water in order to obtain stable enrichment in plasma water of 0.3% during a 60 h period. Enrichment in palmitate of plasma triglycerides (TG) plateaued (0.6-0.76%) whereas plasma cholesterol enrichment increased progressively [0.32 +/- 0.08% (12 h) to 0.78 +/- 0.18% (60 h)]. Endogenous synthesis was estimated to contribute, in post-absorptive subjects, 8-10% of the plasma TG pool and 3-5% of plasma free cholesterol pool. These data agree with results obtained previously using isotope ratio mass spectrometry. The present method will be useful for studies of normal and abnormal lipid metabolism in humans.
CT Cholesterol: AN, analysis
***Cholesterol: BI, biosynthesis**
Cholesterol: BL, blood
Deuterium Oxide: BL, blood
***Deuterium Oxide: ME, metabolism**
Glucose: AN, analysis
Humans
Lactic Acid: AN, analysis
Lactic Acid: BL, blood
Lipids: AN, analysis
***Lipids: BI, biosynthesis**
Lipids: BL, blood
*Mass Fragmentography
Palmitic Acid: AN, analysis
Palmitic Acid: BL, blood
Research Support, Non-U.S. Gov't
Triglycerides: AN, analysis
Triglycerides: BL, blood
RN 50-21-5 (Lactic Acid); 50-99-7 (Glucose); 57-10-3 (Palmitic Acid); 57-88-5 (Cholesterol); 7789-20-0 (Deuterium Oxide)
CN 0 (Lipids); 0 (Triglycerides)

L55 ANSWER 7 OF 37 MEDLINE on STN
AN **97091885** MEDLINE
DN PubMed ID: 8937551
TI Stable isotopes and mass isotopomer study of fatty acid and cholesterol synthesis. A review of the MIDA approach.
AU Lee W N
CS Research and Education Institute, Harbor-UCLA Medical Center, Torrance 90502, USA.
NC R01-DK46353 (NIDDK)
SO Advances in experimental medicine and biology, **(1996)** 399 95-114. Ref: 31
Journal code: 0121103. ISSN: 0065-2598.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals

EM 199703
ED Entered STN: 19970313
Last Updated on STN: 19970313
Entered Medline: 19970303
CT Biopolymers
Carbon Isotopes
***Cholesterol: BI, biosynthesis**
Deuterium
***Fatty Acids: BI, biosynthesis**
***Isotopes**
Mathematics
Research Support, U.S. Gov't, P.H.S.
RN 57-88-5 (Cholesterol); 7782-39-0 (Deuterium)
CN 0 (Biopolymers); 0 (Carbon Isotopes); 0 (Fatty Acids); 0 (Isotopes)

L55 ANSWER 8 OF 37 MEDLINE on STN
AN **96292203** MEDLINE
DN PubMed ID: 8692014
TI In vivo measurement of plasma cholesterol and fatty acid synthesis with deuterated water: determination of the average number of deuterium atoms incorporated.
AU Diraison F; Pachiaudi C; Beylot M
CS INSERM U. 197, Faculte de Medecine Alexis Carrel, Lyon, France.
SO Metabolism: clinical and experimental, **(1996 Jul)** 45 (7) 817-21.
Journal code: 0375267. ISSN: 0026-0495.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199608
ED Entered STN: 19960911
Last Updated on STN: 19960911
Entered Medline: 19960828
AB Fractional lipid synthesis can be measured using the incorporation of deuterium from deuterated water. The calculations require knowledge of the maximum incorporation number (N) of deuterium atoms in the molecules synthesized. For both tissue palmitate and cholesterol, N values have been found to be higher during in vivo versus in vitro experiments. We determined the N values to be used for measuring the fractional synthesis of plasma cholesterol and of palmitate triglycerides (TG). Rats were given drinking water enriched (7% to 10%) with deuterated water, and N was determined from the mass isotopomer distributions of plasma cholesterol and plasma TG palmitate and the deuterium enrichment of plasma water. We found N to be 21 for palmitate and 27 for cholesterol. These values agree with those reported for tissue palmitate and cholesterol in vivo, and are higher than values found in vitro. We also found large deuterium enrichments in plasma glucose and in liver lactate and pyruvate. We suggest that, compared with in vitro studies, in vivo metabolism of these compounds leads to an additional pathway of incorporation of deuterium into lipids through deuterium-labeled acetyl coenzyme A (CoA). This could explain why N values are higher in vivo than in vitro.
CT Check Tags: Male
Acetyl Coenzyme A: ME, metabolism
Animals
Blood Chemical Analysis: MT, methods
Blood Glucose: ME, metabolism
Cholesterol: BI, biosynthesis
*Cholesterol: BL, blood
Deuterium: ME, metabolism
***Deuterium Oxide: ME, metabolism**
***Fatty Acids: BI, biosynthesis**
Fatty Acids: BL, blood
Lactates: ME, metabolism
Lactic Acid
Liver: ME, metabolism
Mass Fragmentography

Palmitates: BL, blood
Pyruvates: ME, metabolism
Pyruvic Acid
Rats
Rats, Sprague-Dawley
Research Support, Non-U.S. Gov't
Triglycerides: BL, blood

RN 127-17-3 (Pyruvic Acid); 50-21-5 (Lactic Acid); 57-88-5 (Cholesterol); 72-89-9 (Acetyl Coenzyme A); 7782-39-0 (Deuterium); 7789-20-0 (Deuterium Oxide)

CN 0 (Blood Glucose); 0 (Fatty Acids); 0 (Lactates); 0 (Palmitates); 0 (Pyruvates); 0 (Triglycerides)

L55 ANSWER 9 OF 37 MEDLINE on STN

AN **94358640** MEDLINE

DN PubMed ID: 8077848

TI Interaction of dietary fat saturation and cholesterol level on cholesterol synthesis measured using deuterium incorporation.

AU Jones P J; Lichtenstein A H; Schaefer E J

CS Division of Human Nutrition, University of British Columbia, Vancouver, Canada.

NC HL39326 (NHLBI)

SO Journal of lipid research, **(1994 Jun)** 35 (6) 1093-101.
Journal code: 0376606. ISSN: 0022-2275.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199410

ED Entered STN: 19941013
Last Updated on STN: 19941013
Entered Medline: 19941003

AB To examine interactive effects of dietary fat saturation and cholesterol level on serum lipids and de novo cholesterogenesis, moderately hypercholesterolemic subjects were fed solid-foods diets containing 30% fat (80 mg C.1000 Kcal-1) in which 2/3 fat was either corn oil or beef tallow, with and without 120 mg C.1000 Kcal-1, for 5 wk. At the end of each diet period, subjects were given deuterium (D) oxide orally and de novo cholesterol synthesis was measured over 24 h from D incorporation into cholesterol as fractional synthesis rates (FSR) and absolute synthetic rate (ASR) into the rapid exchangeable cholesterol pool. Plasma total and low density lipoprotein levels were elevated ($P < 0.01$) with both beef tallow and cholesterol feeding. High density lipoprotein (HDL) levels were not influenced by fat saturation or cholesterol level; however, HDL was higher with addition of cholesterol to corn oil versus beef tallow ($P < 0.02$). Plasma triglycerides were higher ($P < 0.02$) with beef tallow feeding but were not influenced by cholesterol level. FSR was increased ($P < 0.02$) by feeding corn oil, versus beef fat, but not by dietary cholesterol level. Calculated cholesterol pools sizes did not differ across groups; however, ASR was also elevated with corn oil versus beef tallow feeding ($P < 0.02$). Results indicate that corn oil feeding lowers circulating cholesterol by mechanisms other than reduced synthesis, and that cholesterol at the level of supplementation used is not associated with feedback inhibition of cholesterogenesis. However, with the exception of HDL levels, dietary fat saturation and cholesterol levels do not interactively influence circulating lipoprotein cholesterol levels and cholesterol synthesis.

CT Aged
Animals
Cattle
***Cholesterol: BI, biosynthesis**
Cholesterol: BL, blood
*Cholesterol, Dietary: AD, administration & dosage
Corn Oil: AD, administration & dosage
***Deuterium: DU, diagnostic use**
*Dietary Fats: AD, administration & dosage

Fats: AD, administration & dosage
Fatty Acids: AD, administration & dosage
Fatty Acids, Monounsaturated: AD, administration & dosage
Fatty Acids, Unsaturated: AD, administration & dosage
Humans
Lipoproteins, HDL Cholesterol: BL, blood
Lipoproteins, LDL Cholesterol: BL, blood
Meat
Middle Aged
Research Support, Non-U.S. Gov't
Research Support, U.S. Gov't, Non-P.H.S.
Research Support, U.S. Gov't, P.H.S.
Triglycerides: BL, blood

RN 57-88-5 (Cholesterol); 61789-97-7 (tallow); 7782-39-0 (Deuterium); 8001-30-7 (Corn Oil)

CN 0 (Cholesterol, Dietary); 0 (Dietary Fats); 0 (Fats); 0 (Fatty Acids); 0 (Fatty Acids, Monounsaturated); 0 (Fatty Acids, Unsaturated); 0 (Lipoproteins, HDL Cholesterol); 0 (Lipoproteins, LDL Cholesterol); 0 (Triglycerides)

L55 ANSWER 10 OF 37 MEDLINE on STN
AN **93152551** MEDLINE
DN PubMed ID: 8381301
TI Human cholesterol synthesis measurement using deuterated water. Theoretical and procedural considerations.
AU Jones P J; Leitch C A; Li Z C; Connor W E
CS Division of Human Nutrition, University of British Columbia, Vancouver, Canada.
SO Arteriosclerosis and thrombosis : a journal of vascular biology / American Heart Association, **(1993 Feb)** 13 (2) 247-53. Ref: 34
Journal code: 9101388. ISSN: 1049-8834.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199303
ED Entered STN: 19930326
Last Updated on STN: 19930326
Entered Medline: 19930308

AB Human cholesterogenesis is measurable as the rate of incorporation of deuterium derived from deuterium oxide (D2O) within the body water pool into plasma or erythrocyte cholesterol pools. Oral D2O equilibrates across body water, thus enabling extracellular sampling of pools (such as urine) to serve as accurate indicators of intracellular deuterium enrichments at the point of synthesis. Required doses of D2O fall below the threshold associated with negative side effects. Deuterium/carbon incorporation ratios into cholesterol during biosynthesis have been established that are applicable in humans. Models using unconstrained and constrained curve fitting permit improved flexibility in interpretation of deuterium-uptake kinetics. However, sample-size restrictions presently limit the ability of the technique to examine the kinetics within individual lipoprotein species. Correction of enrichment data for proton exchange during combustion and reduction phases of sample preparation is an additional important procedural concern. In summary, the deuterated-water procedure is a useful tool in studies of human cholesterol synthesis that offers the advantages of short measurement interval, relative noninvasiveness, and provision of a direct index of synthesis in comparison with other available techniques.

CT Check Tags: Male
Animals
***Cholesterol: BI, biosynthesis**
***Deuterium: ME, metabolism**
Deuterium Oxide
Humans

Methods
Research Support, Non-U.S. Gov't
Swine
*Water: ME, metabolism
RN 57-88-5 (Cholesterol); 7732-18-5 (Water); 7782-39-0 (Deuterium); 7789-20-0 (Deuterium Oxide)

L55 ANSWER 11 OF 37 MEDLINE on STN
AN **93035907** MEDLINE
DN PubMed ID: 1415685
TI Isotopomer spectral analysis of triglyceride fatty acid synthesis in 3T3-L1 cells.
AU Kharroubi A T; Masterson T M; Aldaghlas T A; Kennedy K A; Kelleher J K
CS Department of Physiology, George Washington University Medical Center, Washington, District of Columbia 20037.
NC AG-00412 (NIA)
GM-33506 (NIGMS)
SO American journal of physiology, (**1992 Oct**) 263 (4 Pt 1) E667-75.
Journal code: 0370511. ISSN: 0002-9513.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199211
ED Entered STN: 19930122
Last Updated on STN: 19980206
Entered Medline: 19921125
AB A new analysis of stable isotope data for biosynthesis reaction, isotopomer spectral analysis (ISA), is demonstrated. ISA is theoretically applicable for polymerization biosynthesis where data are collected using selected ion-monitoring gas chromatography-mass spectrometry. ISA utilizes the discrete spectrum of isotopomer abundances and the multinomial distribution to estimate two key parameters related to the biosynthesis. These parameters are 1) the dilution of the precursor immediately before biosynthesis and 2) the dilution of the newly synthesized product in the sampled compartment. Differentiated 3T3-L1 cells incorporated 2 mM [1,2-13C]acetate into triglyceride palmitate, yielding a spectrum of mass isotopomers of palmitate. The set of equations for the first nine isotopomers were solved for the two parameters using nonlinear regression. We found that precursor dilutions for acetate and glucose were constant over time, whereas the product dilution parameter increased with time, as expected for cells accumulating triglyceride palmitate. Mathematical procedures are presented for calculating 1) the predicted isotopomer fractional abundance values and 2) the correction for atoms other than the tracer atom in the mass ion.
CT 3T3 Cells
Animals
***Carbon Isotopes**
***Fatty Acids: BI, biosynthesis**
Mice
Models, Biological
Myristic Acid
Myristic Acids: ME, metabolism
Palmitates: ME, metabolism
Regression Analysis
Research Support, U.S. Gov't, P.H.S.
*Spectrum Analysis: MT, methods
***Triglycerides: BI, biosynthesis**
RN 544-63-8 (Myristic Acid)
CN 0 (Carbon Isotopes); 0 (Fatty Acids); 0 (Myristic Acids); 0 (Palmitates); 0 (Triglycerides)

L55 ANSWER 12 OF 37 MEDLINE on STN
AN **92044100** MEDLINE
DN PubMed ID: 1940619
TI Measurement of in vivo cholesterol synthesis from 2H2O: a rapid procedure

for the isolation, combustion, and isotopic assay of erythrocyte cholesterol.
AU Wong W W; Hachey D L; Feste A; Leggitt J; Clarke L L; Pond W G; Klein P D
CS Department of Pediatrics, Baylor College of Medicine, Houston, TX.
SO Journal of lipid research, **(1991 Jun)** 32 (6) 1049-56.
Journal code: 0376606. ISSN: 0022-2275.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199112
ED Entered STN: 19920124
Last Updated on STN: 19920124
Entered Medline: 19911204
AB A rapid preparative scale purification of erythrocyte free cholesterol has been developed for measurements of in vivo cholesterol synthesis from 2H2O. The quantity and purity of cholesterol obtained is suitable for combustion, zinc reduction of the water formed, and determination of deuterium isotopic content by gas isotope ratio mass spectrometry. The ability to detect and to quantitate a range of cholesterol synthesis rates is illustrated by measurements on young pigs receiving diets without and with added dietary cholesterol.
CT Animals
***Cholesterol: BI, biosynthesis**
Cholesterol: BL, blood
Cholesterol: IP, isolation & purification
Cholesterol, Dietary: AD, administration & dosage
Chromatography, High Pressure Liquid: MT, methods
***Deuterium**
*Erythrocytes: CH, chemistry
Humans
Research Support, U.S. Gov't, Non-P.H.S.
Spectrum Analysis, Mass: MT, methods
Swine
*Water: CH, chemistry
RN 57-88-5 (Cholesterol); 7732-18-5 (Water); 7782-39-0 (Deuterium)
CN 0 (Cholesterol, Dietary)

L55 ANSWER 13 OF 37 MEDLINE on STN
AN **90345294** MEDLINE
DN PubMed ID: 2200588
TI Use of deuterated water for measurement of short-term cholesterol synthesis in humans.
AU Jones P J
CS Division of Human Nutrition, School of Family and Nutritional Sciences, University of British Columbia, Vancouver, Canada.
SO Canadian journal of physiology and pharmacology, **(1990 Jul)** 68 (7) 955-9. Ref: 24
Journal code: 0372712. ISSN: 0008-4212.
CY Canada
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199009
ED Entered STN: 19901026
Last Updated on STN: 19901026
Entered Medline: 19900920
AB Previous methods for measurement of cholesterol synthesis de novo in humans have either required extended measurement periods or been indirect. Recently, a technique based on the rate of incorporation of deuterium from D2O into the plasma cholesterol pool has been developed. Following oral ingestion of D2O, deuterium enrichment over time in free plasma cholesterol after combustion and reduction was determined using isotope ratio mass spectrometry. This methodology enabled direct measurement of

plasma cholesterol synthesis over intervals as short as 4 h. The technique has been used to demonstrate changes in synthetic rate in response to feeding conditions and genetic influences. Fasting over 36 h resulted in markedly reduced deuterium uptake into cholesterol in healthy males. Diurnal variations in synthetic rate have also been identified, with elevated synthesis observed during nocturnal periods in both fed and fasted subjects. In addition, the influence of apolipoprotein E phenotype on cholesterol synthesis has been shown using this technique. Individuals carrying the apoprotein epsilon 2 allele demonstrated lower synthesis compared with those possessing the epsilon 4 allele. Thus, the deuterium incorporation technique for measuring cholesterol synthesis demonstrates potential as a valuable stable isotope method for human nutrition studies.

CT ***Cholesterol: BI, biosynthesis**
***Deuterium: DU, diagnostic use**
Humans
Research Support, Non-U.S. Gov't

RN 57-88-5 (Cholesterol); 7782-39-0 (Deuterium)

L55 ANSWER 14 OF 37 MEDLINE on STN
AN **86104369** MEDLINE
DN PubMed ID: 3942765
TI Evidence for different isotopic enrichments of acetyl-CoA used for cholesterol synthesis in the liver and intestine: a study in the rat by mass fragmentography after intravenous infusion of [13C]acetate.
AU Ferezou J; Sulpice J C; Lutton C
SO Biochimica et biophysica acta, **(1986 Feb 12)** 875 (2) 227-35.
Journal code: 0217513. ISSN: 0006-3002.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198603
ED Entered STN: 19900321
Last Updated on STN: 19970203
Entered Medline: 19860319

AB Wistar rats were killed 4 h after an intravenous infusion of [1,2-13C]- and [1-14C]acetic acid sodium salt (39 mg, 12.5 microCi/ml, constant rate: 1.2 ml/h). At this time, labeled free cholesterol movements between the organs are still weak and cholesterol labeling in each tissue mainly originates from the in situ incorporation of the exogenous substrate. In male rats, the specific radioactivity of free cholesterol was found to be higher in the intestine (mucosa and wall) than in the liver and plasma. In female and in cholestyramine-fed male rats, cholesterol 14C labeling was close to that of male rats in the intestine, and was markedly higher in the liver. The same variations of 13C excess, calculated by mass fragmentography, indicated that there was no isotopic effect between 13C and 14C precursors. The advantage of this method consisted in obtaining the proportions of labeled molecules according to their molecular weight (M + 1-M + 11) for each sample. Then the distribution of 13C atoms in newly synthesized cholesterol was assessed in each sterogenesis site. In the intestine, about 3/4 of the 13C atoms were found in molecules of weight of at least M + 4 (after incorporation of at least two labeled acetate units). This proportion was only 1/3 in hepatic and plasma free cholesterol. These distinct 13C-labeling patterns clearly indicate that local variations occurred in the isotopic enrichment of acetyl-CoA used for cholesterol formation. Whatever the experimental conditions of this study, cholesterol was synthesized from an acetyl-CoA more 13C enriched in the intestine than in the liver. Such variations probably result from the different dilutions of exogenous acetyl-CoA by the endogenous pool in the liver and intestine. Consequently, the 14C or 13C incorporations measured in the liver and intestinal sterols do not account for absolute rates of cholesterol production by these organs. This study also indicated that after a few hours of infusion, free cholesterol labeling in the plasma originated mainly from cholesterol newly formed in the liver, even when acetate incorporation into cholesterol was higher in the intestine than in the liver.

CT Check Tags: Female; Male
Acetic Acid
Acetic Acids: ME, metabolism
*Acetyl Coenzyme A: ME, metabolism
Animals
***Cholesterol: BI, biosynthesis**
*Intestines: ME, metabolism
***Isotope Labeling**
*Liver: ME, metabolism
Mass Fragmentography
Mathematics
Rats
Rats, Inbred Strains
RN 57-88-5 (Cholesterol); 64-19-7 (Acetic Acid); 72-89-9 (Acetyl Coenzyme A)
CN 0 (Acetic Acids)

L55 ANSWER 15 OF 37 MEDLINE on STN
AN **85258111** MEDLINE
DN PubMed ID: 3894050
TI Effect of drugs, peptide hormones and lipogenic precursors on the relative incorporation of [3H]H2O and carbon into hepatic cholesterol.
AU Bjornsson O G; Pullinger C R; Gibbons G F
SO FEBS letters, **(1985 Aug 5)** 187 (2) 302-6.
Journal code: 0155157. ISSN: 0014-5793.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198509
ED Entered STN: 19900320
Last Updated on STN: 19970203
Entered Medline: 19850924
AB Measurement of the weight of desmosterol produced during its biosynthesis in the presence of tritiated water and triparanol has permitted a direct determination of the relative flux of carbon and tritium (the H/C ratio) into sterol in hepatocytes. The H/C ratio increased with time of incubation irrespective of the nutritional state of the donor animals. This increase was more marked in hepatocytes from starved animals. Pyruvate and lactate increased, and glucagon decreased, the sterol H/C ratio. Addition of pyruvate to incubations containing glucagon resulted in a 32-67% increase in the H/C ratio depending upon nutritional status. Insulin had no effect whilst (-)-hydroxycitrate decreased the ratio by 25%.
CT Check Tags: In Vitro
Animals
*Carbon: ME, metabolism
***Cholesterol: BI, biosynthesis**
Citrates: PD, pharmacology
Fasting
Glucagon: PD, pharmacology
Insulin: PD, pharmacology
Lactates: PD, pharmacology
Lactic Acid
Liver: DE, drug effects
*Liver: ME, metabolism
Pyruvates: PD, pharmacology
Pyruvic Acid
Rats
Research Support, Non-U.S. Gov't
***Tritium: ME, metabolism**
RN 10028-17-8 (Tritium); 11061-68-0 (Insulin); 127-17-3 (Pyruvic Acid); 50-21-5 (Lactic Acid); 57-88-5 (Cholesterol); 6205-14-7 (hydroxycitric acid); 7440-44-0 (Carbon); 9007-92-5 (Glucagon)
CN 0 (Citrates); 0 (Lactates); 0 (Pyruvates)

L55 ANSWER 16 OF 37 MEDLINE on STN

AN 83100402 MEDLINE
DN PubMed ID: 6758778
TI Differential hydrogen exchange during the fatty acid synthetase reaction: deuterium distribution of fatty acids synthesized from [2-2H2]malonyl-CoA.
AU Saito K; Kawaguchi A; Nozoe S; Seyama Y; Okuda S
SO Biochemical and biophysical research communications, (1982 Oct 15) 108 (3) 995-1001.
Journal code: 0372516. ISSN: 0006-291X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198302
ED Entered STN: 19900317
Last Updated on STN: 19900317
Entered Medline: 19830214
CT Brevibacterium
***Deuterium: ME, metabolism**
*Fatty Acid Synthetase Complex: ME, metabolism
***Fatty Acids: BI, biosynthesis**
*Hydrogen: ME, metabolism
Malonyl Coenzyme A: ME, metabolism
Mass Fragmentography
Research Support, Non-U.S. Gov't
Saccharomyces cerevisiae
RN 1333-74-0 (Hydrogen); 524-14-1 (Malonyl Coenzyme A); 7782-39-0 (Deuterium)
CN 0 (Fatty Acids); EC 6.- (Fatty Acid Synthetase Complex)

L55 ANSWER 17 OF 37 MEDLINE on STN
AN 81175008 MEDLINE
DN PubMed ID: 7220494
TI A simple method for the preparation of phosphatidylcholine labelled at 2-acyl position.
AU Connor A M; Brimble P D; Choy P C
SO Preparative biochemistry, (1981) 11 (1) 91-7.
Journal code: 1276634. ISSN: 0032-7484.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198106
ED Entered STN: 19900316
Last Updated on STN: 19900316
Entered Medline: 19810613
AB Phosphatidylcholine with a labelled acyl group at 2 position was prepared enzymatically from lysophosphatidylcholine and labelled fatty acids. Under optimal conditions, over 90% of [5,6,8,9,11,12,14,15-3H] arachidonic acid, [9,10-3H] palmitic acids, [1-14C] linoleic acid or [9,10-3H] oleic acid was incorporated into the phosphatidylcholine, and the incorporation was quantitative up to 500 nmol of [1-14C] linoleic acid. Analysis of the labelled phosphatidylcholine formed indicated that radioactivity was incorporated exclusively at 2-acyl position.
CT Animals
Arachidonic Acids: ME, metabolism
Carbon Radioisotopes
***Isotope Labeling: MT, methods**
Linoleic Acids: ME, metabolism
Microsomes, Liver: ME, metabolism
Oleic Acids: ME, metabolism
Palmitic Acids: ME, metabolism
*Phosphatidylcholines
***Phosphatidylcholines: BI, biosynthesis**
Rats
Research Support, Non-U.S. Gov't
Tritium
RN 10028-17-8 (Tritium)

CN 0 (Arachidonic Acids); 0 (Carbon Radioisotopes); 0 (Linoleic Acids); 0 (Oleic Acids); 0 (Palmitic Acids); 0 (Phosphatidylcholines)

L55 ANSWER 18 OF 37 MEDLINE on STN
AN **77215888** MEDLINE
DN PubMed ID: 327543
TI Mechanism of carbon isotope fractionation associated with lipid synthesis.
AU DeNiro M J; Epstein S
SO Science, **(1977 Jul 15)** 197 (4300) 261-3.
Journal code: 0404511. ISSN: 0036-8075.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197708
ED Entered STN: 19900314
Last Updated on STN: 20010625
Entered Medline: 19770825
AB The low carbon-13/carbon-12 ratio of lipids is shown to result from isotopic fractionation during the oxidation of pyruvate to acetyl coenzyme A. In vitro analysis of the kinetic isotope effects of this reaction indicates that there will be a large, temperature-dependent difference in the carbon-13/carbon-12 ratio between the methyl and carbonyl carbon atoms of acetyl coenzyme A and between those carbon atoms of lipid components which derive from them.
CT Acetates: ME, metabolism
***Carbon Isotopes: ME, metabolism**
*Carboxy-Lyases: ME, metabolism
Escherichia coli: ME, metabolism
Glucose: ME, metabolism
Kinetics
***Lipids: BI, biosynthesis**
*Pyruvate Decarboxylase: ME, metabolism
Pyruvates: ME, metabolism
Research Support, U.S. Gov't, Non-P.H.S.
Saccharomyces cerevisiae: EN, enzymology
RN 50-99-7 (Glucose)
CN 0 (Acetates); 0 (Carbon Isotopes); 0 (Lipids); 0 (Pyruvates); EC 4.1.1. (Carboxy-Lyases); EC 4.1.1.1 (Pyruvate Decarboxylase)

L55 ANSWER 19 OF 37 MEDLINE on STN
AN **77176576** MEDLINE
DN PubMed ID: 1025874
TI [Intensity of incorporation of 2-C14-acetate into the cerebrosides and gangliosides of the brain and spinal cord of rabbits in experimental allergic encephalomyelitis].
Intensivnost' vkliucheniia 2-C14-atsetata v tserebrozidy i gangliozidy golovnogo mozga i spinnogo mozga krolikov pri eksperimental'nom allergicheskom entsefalomielite.
AU Taranova N P; Katsnel'son I P
SO Voprosy meditsinskoi khimii, **(1976 Jan-Feb)** 22 (1) 108-12.
Journal code: 0416601. ISSN: 0042-8809.
CY USSR
DT Journal; Article; (JOURNAL ARTICLE)
LA Russian
FS Priority Journals
EM 197706
ED Entered STN: 19900313
Last Updated on STN: 20000303
Entered Medline: 19770630
AB In rabbits the acute form of experimental allergic encephalomyelitis was caused by inoculation of emulsion of homologous myelin from spinal cord with Freund's adjuvant. In terminal paralytic period of the disease the animals were subcutaneously administered with 2-14C-acetate and a dose 50 micronCi per 100 g of body weight 2 hrs before death. From lumbar and stem sections of the central nervous system purified fractions of

cerebrosides and gangliosides were isolated and their specific radioactivity was determined. The intensity of the cerebrosides synthesis was found to be distinctly decreased not only in the more impaired lumbar region but also in the stem section of the central nervous system, where the centers of demyelinization were not observed. The intensity of the gangliosides synthesis was also markedly decreased, in spite of that their content was not altered in the central nervous system under experimental allergic encephalomyelitis.

CT Check Tags: Male
Acetates: ME, metabolism
Acute Disease
Animals
Brain Chemistry
*Brain Stem: ME, metabolism
***Carbon Radioisotopes: DU, diagnostic use**
***Cerebrosides: BI, biosynthesis**
Cerebrosides: IP, isolation & purification
Chromatography, Thin Layer
*Encephalomyelitis, Autoimmune, Experimental: ME, metabolism
English Abstract
***Gangliosides: BI, biosynthesis**
Gangliosides: IP, isolation & purification
Lumbosacral Region
Rabbits
Spinal Cord: AN, analysis
*Spinal Cord: ME, metabolism

CN 0 (Acetates); 0 (Carbon Radioisotopes); 0 (Cerebrosides); 0 (Gangliosides)

L55 ANSWER 20 OF 37 MEDLINE on STN
AN **77109534** MEDLINE
DN PubMed ID: 835772
TI Enhancement of biliary phospholipid radioactivity by intestinal administration of labeled precursors of hepatic lecithin synthesis.
AU King W 3rd; Tompkins R K
SO American journal of surgery, **(1977 Jan)** 133 (1) 127-33.
Journal code: 0370473. ISSN: 0002-9610.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 197703
ED Entered STN: 19900313
Last Updated on STN: 19900313
Entered Medline: 19770315
AB An animal model is used to study the effects of different radioactive precursors of lecithin, administered intestinally, upon biliary lecithin synthesis. Choline caused greater biliary lecithin radioactivity than choline-labeled lecithin or palmitic acid. The clinical application of this finding is discussed with relation to patients with gallstones or potentially lithogenic bile. Oral administration of choline may increase biliary lecithin concentration and, thus, cholesterol-holding capacity in this patient group, thereby changing the character of the bile to a non-lithogenic state.

CT Animals
*Bile Acids and Salts: ME, metabolism
***Carbon Radioisotopes: ME, metabolism**
Cholelithiasis: ET, etiology
*Choline: PD, pharmacology
Injections
Intestines
*Liver: ME, metabolism
Models, Biological
Palmitic Acids: AD, administration & dosage
*Palmitic Acids: PD, pharmacology
Phosphatidylcholines: BI, biosynthesis
***Phospholipids: BI, biosynthesis**

Swine
RN 62-49-7 (Choline)
CN 0 (Bile Acids and Salts); 0 (Carbon Radioisotopes); 0 (Palmitic Acids); 0 (Phosphatidylcholines); 0 (Phospholipids)

L55 ANSWER 21 OF 37 MEDLINE on STN
AN **74062356** MEDLINE
DN PubMed ID: 4587725
TI An efficient biosynthetic method to prepare fatty acyl chains highly enriched with 13C.
AU Cronan J E Jr; Batchelor J G
SO Chemistry and physics of lipids, **(1973 Oct)** 11 (3) 196-202.
Journal code: 0067206. ISSN: 0009-3084.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197402
ED Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19740222
CT Acetates: ME, metabolism
***Carbon Isotopes**
Carbon Radioisotopes
Citrate (si)-Synthase
Escherichia coli: ME, metabolism
Evaluation Studies
***Fatty Acids: BI, biosynthesis**
Isotope Labeling
Magnetic Resonance Spectroscopy
Methods
Mutation
Transduction, Genetic
CN 0 (Acetates); 0 (Carbon Isotopes); 0 (Carbon Radioisotopes); 0 (Fatty Acids); EC 4.1.3.7 (Citrate (si)-Synthase)

L55 ANSWER 22 OF 37 MEDLINE on STN
AN **73216685** MEDLINE
DN PubMed ID: 4717997
TI Labeling plasma lipoproteins with radioactive cholesterol.
AU Sodhi H S; Kudchodkar B J
SO Journal of laboratory and clinical medicine, **(1973 Jul)** 82 (1) 111-24.
Journal code: 0375375. ISSN: 0022-2143.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 197309
ED Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19730913
CT Check Tags: Female; Male
Acetates: AD, administration & dosage
Administration, Oral
Adult
Blood Protein Disorders: BL, blood
Carbon Isotopes
Cholesterol: AD, administration & dosage
Cholesterol: AN, analysis
Cholesterol: BI, biosynthesis
*****Cholesterol: BL, blood
Cholesterol: ME, metabolism
Ethanol: AD, administration & dosage
Feces: AN, analysis
Humans

Hyperlipidemia: BL, blood
Injections, Intravenous
***Isotope Labeling**
Lipoproteins: BI, biosynthesis
*Lipoproteins: BL, blood
Methods
Mevalonic Acid: AD, administration & dosage
Middle Aged
Tritium
Xanthomatosis: BL, blood
RN 10028-17-8 (Tritium); 150-97-0 (Mevalonic Acid); 57-88-5 (Cholesterol); 64-17-5 (Ethanol)
CN 0 (Acetates); 0 (Carbon Isotopes); 0 (Lipoproteins)

L55 ANSWER 23 OF 37 MEDLINE on STN
AN **73145444** MEDLINE
DN PubMed ID: 4735044
TI A simple biosynthetic method for the preparation of glycerol-labelled phosphatidylcholine.
AU Parthasarathy S; Ganguly J
SO Biochimica et biophysica acta, **(1973 Jan 19)** 296 (1) 62-4.
Journal code: 0217513. ISSN: 0006-3002.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197305
ED Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19730518
CT ***Carbon Isotopes**
Chromatography, Thin Layer
Darkness
Glycerides: ME, metabolism
*Glycerol
Glycerol: ME, metabolism
Isotope Labeling
Methods
***Phosphatidylcholines: BI, biosynthesis**
Phosphatidylcholines: IP, isolation & purification
Phospholipids: ME, metabolism
Plants: ME, metabolism
Seeds: ME, metabolism
Soybeans
RN 56-81-5 (Glycerol)
CN 0 (Carbon Isotopes); 0 (Glycerides); 0 (Phosphatidylcholines); 0 (Phospholipids)

L55 ANSWER 24 OF 37 MEDLINE on STN
AN **72098476** MEDLINE
DN PubMed ID: 5167797
TI [Use of different C 14 precursors for the biosynthesis of labelled fucidin].
Ispol'zovanie rallichnykh C 14 -predshestvennikov dlia biosinteza mechenogo fuzidina.
AU Grianova N S; Afonin V I; Sazykin Iu O
SO Antibiotiki, **(1971 Sep)** 16 (9) 775-7.
Journal code: 0375020. ISSN: 0003-5637.
CY USSR
DT Journal; Article; (JOURNAL ARTICLE)
LA Russian
FS Priority Journals
EM 197204
ED Entered STN: 19900310
Last Updated on STN: 19990129
Entered Medline: 19720407

CT Check Tags: Comparative Study
Acetates: ME, metabolism
Acetic Acids: ME, metabolism
***Carbon Isotopes**
Culture Media
Fermentation
***Fusidic Acid: BI, biosynthesis**
Glucose: ME, metabolism
Mitosporic Fungi: ME, metabolism
Radioactivity
Sodium: ME, metabolism
RN 50-99-7 (Glucose); 6990-06-3 (Fusidic Acid); 7440-23-5 (Sodium)
CN 0 (Acetates); 0 (Acetic Acids); 0 (Carbon Isotopes); 0 (Culture Media)

L55 ANSWER 25 OF 37 MEDLINE on STN
AN **71288112** MEDLINE
DN PubMed ID: 4398281
TI [A method for preparing radioactive labeled phosphatides using soybeans in vivo].
Uber eine in-vivo-Methode zur Herstellung radioaktiv markerter Phosphatide mit Sojabohnen.
AU Holzl J; Wagner H
SO Zeitschrift fur Naturforschung. Teil B: Chemie, Biochemie, Biophysik, Biologie, **(1971 May)** 26 (5) 425-34.
Journal code: 0364315. ISSN: 0044-3174.
CY GERMANY, WEST: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA German
FS Priority Journals
EM 197111
ED Entered STN: 19900101
Last Updated on STN: 19980206
Entered Medline: 19711112
CT Acetates: ME, metabolism
Carbon Isotopes
Choline: ME, metabolism
Fatty Acids: ME, metabolism
Glycerol: ME, metabolism
Methods
Phosphates: ME, metabolism
Phosphatidylcholines: BI, biosynthesis
Phosphatidylethanolamines: BI, biosynthesis
***Phospholipids: BI, biosynthesis**
Phosphorus Isotopes
Radiation Effects
***Radioisotopes**
Radiometry
*Soybeans
RN 56-81-5 (Glycerol); 62-49-7 (Choline)
CN 0 (Acetates); 0 (Carbon Isotopes); 0 (Fatty Acids); 0 (Phosphates); 0 (Phosphatidylcholines); 0 (Phosphatidylethanolamines); 0 (Phospholipids); 0 (Phosphorus Isotopes); 0 (Radioisotopes)

L55 ANSWER 26 OF 37 MEDLINE on STN
AN **71090573** MEDLINE
DN PubMed ID: 4322218
TI Incorporation of 32P-orthophosphate into phospholipids by a toxigenic and a nontoxigenic strain of Aspergillus flavus.
AU Gupta S R; Viswanathan L; Venkitasubramanian T A
SO Mycopathologia et mycologia applicata, **(1970 Dec 28)** 42 (1) 137-44.
Journal code: 7505688. ISSN: 0027-5530.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals

EM 197103
ED Entered STN: 19900101
Last Updated on STN: 19900101
Entered Medline: 19710312
CT Check Tags: Comparative Study
*Aflatoxins: BI, biosynthesis
Aspergillus: AN, analysis
Aspergillus: GD, growth & development
*Aspergillus: ME, metabolism
Chromatography, Thin Layer
Culture Media
Gels
Glucose
*Phosphates: ME, metabolism
Phosphatidylcholines: BI, biosynthesis
Phosphatidylethanolamines: BI, biosynthesis
Phosphatidylinositols: BI, biosynthesis
***Phospholipids: BI, biosynthesis**
Phosphorus: AN, analysis
***Phosphorus Isotopes**
Saccharomyces
Silicon Dioxide
Species Specificity
Sucrose
RN 50-99-7 (Glucose); 57-50-1 (Sucrose); 7631-86-9 (Silicon Dioxide); 7723-14-0 (Phosphorus)
CN 0 (Aflatoxins); 0 (Culture Media); 0 (Gels); 0 (Phosphates); 0 (Phosphatidylcholines); 0 (Phosphatidylethanolamines); 0 (Phosphatidylinositols); 0 (Phospholipids); 0 (Phosphorus Isotopes)

L55 ANSWER 27 OF 37 MEDLINE on STN
AN **68157948** MEDLINE
DN PubMed ID: 5237893
TI On the constancy of deuterium fractionation in the biosynthesis of fatty acids since the miocene period.
AU Zborowski G; Ponticorvo L; Rittenberg D
SO Proceedings of the National Academy of Sciences of the United States of America, **(1967 Oct)** 58 (4) 1660-3.
Journal code: 7505876. ISSN: 0027-8424.
Report No.: NASA-68157948.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Space Life Sciences
EM 196805
ED Entered STN: 19900101
Last Updated on STN: 19900101
Entered Medline: 19680501
CT Amino Acids: AN, analysis
Animals
*Biogenesis
California
Chromatography, Gas
***Deuterium: AN, analysis**
Fatty Acids: AN, analysis
***Fatty Acids: BI, biosynthesis**
Fishes
Geology
Lipids: AN, analysis
Rats
West Indies
RN 7782-39-0 (Deuterium)
CN 0 (Amino Acids); 0 (Fatty Acids); 0 (Lipids)

L55 ANSWER 28 OF 37 MEDLINE on STN
AN **67216680** MEDLINE

DN PubMed ID: 5974909
TI Fatty acid synthesis and gluconeogenesis in rats fed a high caloric diet.
AU Shigeta Y; Oji N; Hoshi M; Kang M
SO Metabolism: clinical and experimental, **(1966 Aug)** 15 (8) 761-6.
Journal code: 0375267. ISSN: 0026-0495.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 196710
ED Entered STN: 19900101
Last Updated on STN: 19900101
Entered Medline: 19671020
CT Check Tags: In Vitro
*Acetates: ME, metabolism
Alanine: ME, metabolism
Animals
Blood Glucose: BI, biosynthesis
Carbon Dioxide
***Carbon Isotopes: ME, metabolism**
*Diet
***Fatty Acids: BI, biosynthesis**
*Gluconeogenesis
Liver Glycogen: BI, biosynthesis
Rats
RN 124-38-9 (Carbon Dioxide); 56-41-7 (Alanine)
CN 0 (Acetates); 0 (Blood Glucose); 0 (Carbon Isotopes); 0 (Fatty Acids); 0 (Liver Glycogen)

L55 ANSWER 29 OF 37 MEDLINE on STN
AN **67202029** MEDLINE
DN PubMed ID: 4291569
TI The distribution of tritium in fatty acids synthesized from tritiated glucose and tritiated water by rat adipose tissue.
AU Foster D W; Katz J
SO Biochimica et biophysica acta, **(1966 Dec 7)** 125 (3) 422-7.
Journal code: 0217513. ISSN: 0006-3002.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 196709
ED Entered STN: 19900101
Last Updated on STN: 19900101
Entered Medline: 19670925
CT Check Tags: Male
*Adipose Tissue: ME, metabolism
Animals
Carbon Isotopes
Epididymis
***Fatty Acids: BI, biosynthesis**
*Glucose: ME, metabolism
*Hydrogen: ME, metabolism
*NAD: ME, metabolism
Palmitic Acids: BI, biosynthesis
Rats
Stearic Acids: BI, biosynthesis
***Tritium: AN, analysis**
Water
RN 10028-17-8 (Tritium); 1333-74-0 (Hydrogen); 50-99-7 (Glucose); 53-84-9 (NAD); 7732-18-5 (Water)
CN 0 (Carbon Isotopes); 0 (Fatty Acids); 0 (Palmitic Acids); 0 (Stearic Acids)

L55 ANSWER 30 OF 37 MEDLINE on STN
AN **67179526** MEDLINE

DN PubMed ID: 5971049
TI Sites of control of hepatic cholesterol biosynthesis.
AU Gould R G; Swyryd E A
SO Journal of lipid research, (1966 Sep) 7 (5) 698-707.
Journal code: 0376606. ISSN: 0022-2275.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 196709
ED Entered STN: 19900101
Last Updated on STN: 19900101
Entered Medline: 19670907
AB An inhibition in the conversion of mevalonate to cholesterol has been demonstrated in liver of cholesterol-fed rats by both in vitro and in vivo methods. Synthesis decreased to 30% of the control value after 1 week and 20% after 1 month on a 1% cholesterol diet. After a year, synthesis from mevalonate was almost completely inhibited. The rate of conversion of squalene to cholesterol was not consistently decreased but that of farnesyl pyrophosphate to cholesterol was decreased considerably. The rate of conversion of mevalonate to farnesyl pyrophosphate by a soluble liver enzyme preparation was also decreased in cholesterol-fed animals. Sites of inhibition of cholesterol synthesis were detected before mevalonate, between mevalonate and farnesyl pyrophosphate, and after farnesyl pyrophosphate, probably at the conversion of farnesyl pyrophosphate to squalene. The inhibition of mevalonate conversion to cholesterol developed more slowly than that of acetate and appeared to be secondary to it. The maximum capacities of normal liver homogenates and slices to synthesize cholesterol from mevalonate were shown to be far greater than from acetate. Consequently, sites of inhibition after mevalonate probably do not have a significant effect on the over-all rate of cholesterol synthesis in the intact cholesterol-fed animal.
CT Check Tags: Male
Animals
***Carbon Isotopes**
Cholesterol: AN, analysis
***Cholesterol: BI, biosynthesis**
Liver: AN, analysis
*Liver: ME, metabolism
Mevalonic Acid: ME, metabolism
Rats
RN 150-97-0 (Mevalonic Acid); 57-88-5 (Cholesterol)
CN 0 (Carbon Isotopes)

L55 ANSWER 31 OF 37 MEDLINE on STN
AN **67160888** MEDLINE
DN PubMed ID: 5965303
TI Simultaneous P32- and C14-labeling of phospholipids by germinating soybeans.
AU Hoelzl J; Wagner H
SO Journal of lipid research, (1966 Jul) 7 (4) 569-70.
Journal code: 0376606. ISSN: 0022-2275.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 196707
ED Entered STN: 19900101
Last Updated on STN: 19900101
Entered Medline: 19670721
AB Doubly labeled ((32)P and (14)C) phospholipids of high specific activity are obtained by incorporating the labels during germination of soybeans.
CT Check Tags: In Vitro
Acetates: ME, metabolism
***Carbon Isotopes: ME, metabolism**
Phosphates: ME, metabolism

***Phospholipids: BI, biosynthesis**
***Phosphorus Isotopes: ME, metabolism**
*Plants, Edible: ME, metabolism
CN 0 (Acetates); 0 (Carbon Isotopes); 0 (Phosphates); 0 (Phospholipids); 0 (Phosphorus Isotopes)

L55 ANSWER 32 OF 37 MEDLINE on STN
AN **67126401** MEDLINE
DN PubMed ID: 4289988
TI Effect of TSH, acetylcholine, epinephrine, serotonin and synkavite on 32-P incorporation into phospholipids in dog-thyroid slices.
AU Altman M; Oka H; Field J B
SO Biochimica et biophysica acta, **(1966 Jun 1)** 116 (3) 586-8.
Journal code: 0217513. ISSN: 0006-3002.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 196705
ED Entered STN: 19900101
Last Updated on STN: 19900101
Entered Medline: 19670522
CT Check Tags: In Vitro
*Acetylcholine: PD, pharmacology
Animals
Atropine: PD, pharmacology
Dogs
*Epinephrine: PD, pharmacology
Phosphatidylcholines: BI, biosynthesis
Phosphatidylethanolamines: BI, biosynthesis
Phosphatidylinositols: BI, biosynthesis
***Phospholipids: BI, biosynthesis**
***Phosphorus Isotopes: ME, metabolism**
Puromycin: PD, pharmacology
*Serotonin: PD, pharmacology
*Thyroid Gland: DE, drug effects
*Thyroid Gland: ME, metabolism
*Thyrotropin: PD, pharmacology
*Vitamin K: PD, pharmacology
RN 12001-79-5 (Vitamin K); 50-67-9 (Serotonin); 51-43-4 (Epinephrine); 51-55-8 (Atropine); 51-84-3 (Acetylcholine); 53-79-2 (Puromycin); 9002-71-5 (Thyrotropin)
CN 0 (Phosphatidylcholines); 0 (Phosphatidylethanolamines); 0 (Phosphatidylinositols); 0 (Phospholipids); 0 (Phosphorus Isotopes)

L55 ANSWER 33 OF 37 MEDLINE on STN
AN **67061525** MEDLINE
DN PubMed ID: 6016079
TI Iodide transport: inhibition by agents reacting at the membrane.
AU Larsen P R; Wolff J
SO Science, **(1967 Jan 20)** 155 (760) 335-6.
Journal code: 0404511. ISSN: 0036-8075.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 196703
ED Entered STN: 19900101
Last Updated on STN: 19900101
Entered Medline: 19670310
CT Check Tags: In Vitro
Animals
*Antibiotics, Antifungal: PD, pharmacology
Cattle
*Iodides: ME, metabolism
*Lysophosphatidylcholines: PD, pharmacology

*Phospholipases: PD, pharmacology
***Phospholipids: BI, biosynthesis**
***Phosphorus Isotopes: ME, metabolism**
*Potassium: ME, metabolism
*Thyroid Gland: ME, metabolism
*Thyrotropin: PD, pharmacology
*Venoms: PD, pharmacology
RN 7440-09-7 (Potassium); 9002-71-5 (Thyrotropin)
CN 0 (Antibiotics, Antifungal); 0 (Iodides); 0 (Lysophosphatidylcholines); 0 (Phospholipids); 0 (Phosphorus Isotopes); 0 (Venoms); EC 3.1.- (Phospholipases)

L55 ANSWER 34 OF 37 MEDLINE on STN
AN **67004390** MEDLINE
DN PubMed ID: 4287973
TI Studies on brain phospholipids. IV. Post-mortem breakdown of phosphoinositides and phosphatidic acid, and P32-incorporation into phospholipids in various states of the tissue.
AU Hayashi K; Yagihara Y; Nakamura I; Yamazoe S
SO Journal of biochemistry, **(1966 Jul)** 60 (1) 42-51.
Journal code: 0376600. ISSN: 0021-924X.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 196611
ED Entered STN: 19900101
Last Updated on STN: 19900101
Entered Medline: 19661129
CT Check Tags: In Vitro
Animals
*Brain: ME, metabolism
Chemistry, Analytical
Chromatography
Guinea Pigs
Ouabain: PD, pharmacology
*Phosphatidylinositols: ME, metabolism
***Phospholipids: BI, biosynthesis**
***Phosphorus Isotopes: ME, metabolism**
Sodium: PD, pharmacology
RN 630-60-4 (Ouabain); 7440-23-5 (Sodium)
CN 0 (Phosphatidylinositols); 0 (Phospholipids); 0 (Phosphorus Isotopes)

L55 ANSWER 35 OF 37 MEDLINE on STN
AN **67003285** MEDLINE
DN PubMed ID: 5912364
TI Perfusion in situ with tritium oxide to measure hepatic lipogenesis and lipid secretion. Normal and orotic acid-fed rats.
AU Windmueller H G; Spaeth A E
SO Journal of biological chemistry, **(1966 Jun 25)** 241 (12) 2891-9.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 196611
ED Entered STN: 19900101
Last Updated on STN: 19900101
Entered Medline: 19661127
CT Animals
***Cholesterol: BI, biosynthesis**
***Fatty Acids: BI, biosynthesis**
Hyperlipidemia: CI, chemically induced
***Lipids: BI, biosynthesis**
*Liver: ME, metabolism
*Orotic Acid: PD, pharmacology

Perfusion
Rats
Surface-Active Agents: PD, pharmacology
Triglycerides: BL, blood
***Tritium: ME, metabolism**
RN 10028-17-8 (Tritium); 57-88-5 (Cholesterol); 65-86-1 (Orotic Acid)
CN 0 (Fatty Acids); 0 (Lipids); 0 (Surface-Active Agents); 0 (Triglycerides)

L55 ANSWER 36 OF 37 MEDLINE on STN
AN **66157647** MEDLINE
DN PubMed ID: 5940930
TI Phospholipid metabolism in kidney. 3. Biosynthesis of phospholipids from radioactive precursors in rabbit renal cortex slices.
AU Tinker D O; Hanahan D J
SO Biochemistry, **(1966 Feb)** 5 (2) 423-35.
Journal code: 0370623. ISSN: 0006-2960.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 196610
ED Entered STN: 19900101
Last Updated on STN: 19900101
Entered Medline: 19661001
CT Check Tags: In Vitro
Animals
Chromatography, Thin Layer
Cyanides: PD, pharmacology
*Glucose: ME, metabolism
*Kidney: ME, metabolism
*Linoleic Acids: ME, metabolism
Ouabain: PD, pharmacology
*Palmitic Acids: ME, metabolism
Phosphatidylcholines: BI, biosynthesis
Phosphatidylethanolamines: BI, biosynthesis
***Phospholipids: BI, biosynthesis**
***Phosphorus Isotopes: ME, metabolism**
Rabbits
*Serine: ME, metabolism
Sphingomyelins: BI, biosynthesis
RN 50-99-7 (Glucose); 56-45-1 (Serine); 630-60-4 (Ouabain)
CN 0 (Cyanides); 0 (Linoleic Acids); 0 (Palmitic Acids); 0 (Phosphatidylcholines); 0 (Phosphatidylethanolamines); 0 (Phospholipids); 0 (Phosphorus Isotopes); 0 (Sphingomyelins)

L55 ANSWER 37 OF 37 MEDLINE on STN
AN **66071506** MEDLINE
DN PubMed ID: 4285354
TI Biochemical changes in denervated skeletal muscle. 3. The effect of denervation atrophy on the concentration and 32P labelling of the individual phospholipids of the rat gastrocnemius.
AU Graff G L; Hudson A J; Strickland K P
SO Biochimica et biophysica acta, **(1965 Jul 8)** 104 (2) 543-53.
Journal code: 0217513. ISSN: 0006-3002.
Report No.: NASA-66071506.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Space Life Sciences
EM 196603
ED Entered STN: 19900101
Last Updated on STN: 19900101
Entered Medline: 19660326
CT Check Tags: In Vitro
Animals
*Muscle Denervation

*Muscles: ME, metabolism
*Muscular Atrophy: ME, metabolism
Phosphatidylinositols: BI, biosynthesis
***Phospholipids: BI, biosynthesis**
***Phosphorus Isotopes: ME, metabolism**
Rats
CN 0 (Phosphatidylinositols); 0 (Phospholipids); 0 (Phosphorus Isotopes)

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